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POLIOMYELITIS.[1]

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IT has taken some time to decide the exact scope of this lecture, and I have concluded that the primary object is to give what little help we can in the matter of early diagnosis to those who are seeing these cases early or who presumably will be seeing them in the near future. This talk will be entirely clinical and will deal with symptoms, signs and diagnosis, and the endeavour has been made to make it as concise as possible. I ask you to tolerate generously the disorderly nature of my paper; unlike the cases under discussion, it has had an incubation period of only seven days, and those days, as you may guess, have not been free from interruption.

[1] Read at a special meeting of the Victorian Branch of the British Medical Association on July 2, 1937.

Symptoms.

For many years two distinct phases of the disease have been manifest, namely, the febrile phase and the paralytic phase. It has long been recognized that the severity of both phases and their relations to each other have been extremely variable, producing a series of clinical types of the disease. To enumerate briefly the symptoms of the febrile phase: we have fever, irritability, anorexia, drowsiness, headache, vomiting, pains in the back and back of the neck and limbs, and hyperæsthesia of the back and along the large nerve trunks, all in variable degree. There are six main variations of this phase. They are indicated in the following schematic representation.

Group I.—Group I represents the very mild undiagnosable febrile phase probably suffered by most of us at some time without any sequelæ, but leaving a lasting immunity.

Group II.—Group II represents a variety of which you have all had experience at some time. The febrile phase is mild, and the disease is usually unrecognized until some few weeks later, when the child is brought in because of a limp or a weakness

in one arm. Such a case has actually occurred intermediary between two severe cases in the present epidemic.

Group III.—Group III represents abortive poliomyelitis. Here the febrile phase is mild, but usually noticeable, occurs during an epidemic, and its nature is recognizable only because of that, there being practically no spinal symptoms or signs and no resulting paralysis. Experimentally the sera in such cases have been found to have many times the neutralizing power of the serum of badly paralysed patients. This may be of importance in the future of serum treatment.

Group IV.—The fourth group depicts Draper's dromedary type of poliomyelitis. Here we have a mild febrile phase, which subsides for a few days and then recurs in greater intensity, to be followed within a day or so by the paralytic phase. There have been several cases of this type in the present epidemic, and one child at least attended school between the two humps of her dromedary.[1]

Group V.—The fifth group is by far the commonest clinical type seen in this epidemic, namely, the abrupt, well-marked febrile phase, going on very quickly, one may say unusually quickly, to the paralytic phase.

Group VI.—Group VI depicts a comparative rarity in this epidemic, namely, non-paralytic poliomyelitis, in which, following a well-marked febrile phase with all the spinal signs and symptoms pronounced, no paralytic phase eventuates. The serum in these cases has been shown experimentally to be intermediate in neutralizing power between the sera in cases of paralytic and abortive types. I use the word "experimentally" with definite intent, and it is interesting to note that Jensen, who conducted these experiments, has now refuted his earlier conclusions.

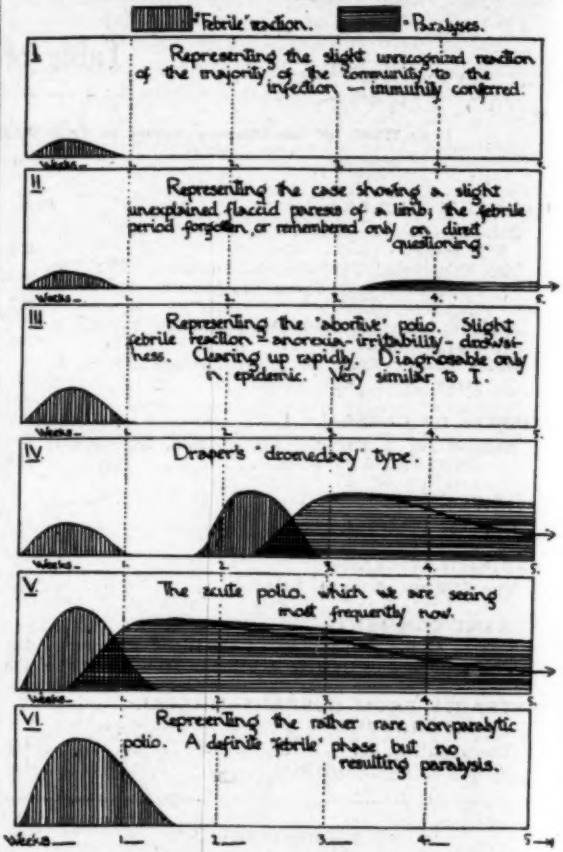
The symptoms are given here in approximately the order in which they have appeared, and a very rough estimate is given of their frequency in the epidemic to date.

Fever.

Fever invariably occurred. It is usually sudden in onset, attacking an apparently perfectly well child. In at least four cases the child has awakened during the night, feverish, flushed and restless, with some headache, but has gone off to sleep again. The temperature has tended to be high—38·9° to 39·4° C. (102° to 103° F.)—taking a week or more to settle down slowly by lysis; fever in some cases has been lower than this. In the dromedary cases the fever has subsided for two or three days, then returned and become higher than before. Often, too, the febrile aspect of the first hump is missed altogether.

Drowsiness.

Drowsiness has also been quite constant; but it is of a particular type. The parent will volunteer the symptom, the ward sister will corroborate this; but whenever the doctor wishes to examine the child or take his photograph he appears alert, lucid and cooperative. We have used this type of drowsiness as a valuable aid to diagnosis; but it is from



necessity an observation of the nurse or mother rather than the doctor. One child with marked spine signs and symptoms and with early respiratory involvement conducted the following conversation with me:

"Doctor", she said "have you a car?"

"Yes", I replied.

"Then would you drive me back to see my mummy in Chelsea?"

That is typical of the lucidity of these children, even when in desperate straits.

Another example is that of a boy, aged ten years, with a temperature of 38·6° C. (101·5° F.), a good deal of spine and neck stiffness, and a right facial weakness, who said: "How many cases are there now? Twenty-three, isn't it? Oh, no, it will be 24 now; I'll be the twenty-fourth."

Only one patient in the epidemic so far, that is, in about 60 cases, has been so stuporose as to be unable to answer questions.

Headache.

The sudden onset of frontal headache in association with fever in a perfectly well child has been very common. If the child volunteers the infor-

[1] As the epidemic goes on more and more of these dromedary types are seen. They seem to be matching Group V in frequency. In some, too, there is a continuity between the two humps, the patient being vaguely ill for seven or eight days, sometimes as long as thirteen days, as in the "straggling" types in other epidemics.

mation that he has a headache or if the mother says that the child "was crying with headache" and was indicating the frontal region with his hand, then this is a very suggestive symptom. Frontal headache has been present in about 80% of cases; its complete absence is rather against the diagnosis.

Vomiting.

Somewhere in the history there has usually been an attack of nausea, with or without vomiting, the majority actually vomiting once or twice only. In isolated cases only, vomiting has been repeated five or six times. Children vomit so easily that we have not regarded it as of great help in diagnosis, particularly as it has sometimes followed a purgative. In association with other suggestive features, however, it has its place in diagnosis. Diarrhœa has, very strikingly, not been a feature of this epidemic. The patients with abdominal pain have in several instances been given purgatives previously.

Anorexia.

Anorexia has been practically constant, but, of course, is a very common feature of all the acute fevers of childhood, and as a diagnostic sign has obvious limitations. A good appetite is against the diagnosis, however.

Pain in the Back of the Neck.

Pain in the back of the neck has been a spontaneous symptom in at least 90% of the cases. Pain in the back, sometimes quite low, has occurred slightly less frequently. It is of great importance that the existence of these symptoms be voluntarily mentioned by the child. Anxious parents are suggesting rather frequently to children the presence of pain in the back of the neck, with disastrous results from the point of view of assessing its worth in diagnosis. Frequently in the early stages this symptom is present only when an attempt is made to elicit the spinal signs; its presence then, again mentioned spontaneously, is of great importance.

Disturbance of Micturition.

Disturbance of micturition has been an interesting symptom. There has often been a complaint by the parents that difficulty has been experienced in commencing the act, and in hospital there have been several children with distension of the bladder to the umbilicus during their stay in hospital. Often a few ounces are passed; but by percussion there is found still a good deal of urine present. A hot bath has been the most satisfactory means of producing the complete act. There seems some sensory impairment also, for there is never a complaint of the pain of a full bladder. It is possible that some œdema of the cord producing temporary mild pyramidal and posterior column embarrassment is responsible for the major part of this symptom.

Muscle Pain and Hyperæsthesia.

Muscle pain and hyperæsthesia have been surprisingly rare; in other epidemics they have been quite a marked feature. Abdominal pain has been complained of by some of the children; it has been produced by purgatives in at least some of the cases. Sometimes it may indicate early involvement of abdominal groups of muscles.

Weakness of Muscles.

Weakness of a limb or other muscle groups as a diagnostic feature is, of course, invaluable, but from the point of view of early diagnosis it is not so helpful. However, one of the patients now in a respirator complained of some weakness twenty-four hours from the onset of the disease, and I have seen two cases with facial paresis on the first examination, one, in addition, having a hypoglossal and unilateral palatal weakness.

Tremor.

Slight fine tremor is a common accompaniment of many of the severe febrile disturbances of childhood. Parents do not usually mention it as a symptom. But the tremor of poliomyelitis is gross enough to attract the parents' attention and cause them to mention it as "the shakes" or "he was so trembly in the arms that he could not hold a glass of water". A description like that, volunteered by the parent, is almost diagnostic. The tremor is of the "intention" type seen in disseminated sclerosis. Often the tremor is unilateral or asymmetrical in intensity; its diagnostic value is then enhanced. It often precedes paralysis in a limb.

Signs on Examination.

General Appearances.

Almost without exception the patients are well-developed children. There is a marked flush and the temperature is from 38·9° to 39·5° C. (100° to 103° F.). The pulse rate is distinctly high, higher than a proportionate rate, and it is quite common to see a child of four years with a temperature of 38·4° C. (101° F.) and a pulse rate of 140 to 150 per minute. Again I must repeat that the patients are perfectly lucid and helpful, and lie quietly in bed. They breathe quietly, they have no cough or coryza, and if they have any pain they certainly make very little fuss about it. They develop a strangely staid demeanour for their age, particularly the ones going on to extensive paralysis. They talk in monosyllables, if at all. They appear to realize that they may have a fight ahead of them, and are conserving all energy in preparation for this. If on entering a house one can hear screams, coughs or tantrums, one can feel fairly sure that the case is not poliomyelitis. The disease is intensely "organic". The child who giggles and says, "Oh, doctor, I do hope I haven't got it", probably has not.

Photophobia has been quite rare; the boy depicted in the cinematograph film had it quite markedly. Nystagmus has been observed by me in only three cases. One boy had diplopia on looking up and to the right.

The nose is usually singularly clean.

Quite a high proportion of patients have had injected throats, but nothing much more than this.

A heavily coated tongue has been quite a feature of the cases so far.

The Spinal Group of Signs.

The spinal signs have been the mainstay of diagnosis, and it is extremely difficult to make a positive diagnosis in their complete absence.

Neck Stiffness.—Neck stiffness without head retraction is extremely important. There are very few diseases in which the patients have such a marked degree of neck stiffness and still lie apparently comfortably without the head retracted. In testing for this sign it is most important that the flexing be done very slowly and gently. Vigorous flexion will produce pain and resentment in any of the acute fevers. In some cases it is better just to sit the patient up and hold a penny or a torch in the region of the umbilicus, and ask him to look at it. The sudden halt in the neck flexion, accompanied by a momentary frown or grunt of pain, is very suggestive. The eyes are willing but the head is not.

Rigidity.—Rigidity of the back is usually an extension of the neck rigidity. It is often to be observed when the neck is being flexed that the shoulders and the back come up in one piece, the child complaining that it hurts down the back. They cannot kiss their knees without great effort, and usually give a grunt of pain. If asked to pick up an object from the floor, they go down with flexed knees but with extended, rigid back and neck.

Amoss's Sign.—Amoss's sign is due largely to weakness in the trunk muscles. It is elicited by asking or assisting them to sit up, when it is found that they cannot do so without propping themselves in a tripod fashion with their arms behind them. This sign has been almost completely useless as an early sign of the disease.

The Head Lag.—Head lag has proved of great value in the early diagnosis. It is due to a curious weakness of the anterior group of neck muscles and may be present within twelve hours of the onset. When present it is a definite phenomenon. If both shoulders of the patient are slowly raised off the bed, the examiner facing the patient, the head, instead of readily maintaining its normal relation to the shoulders, lags heavily back until, with an obviously severe effort, which sometimes produces a momentary *risus sardonicus* from platysmal contraction, the patient jerks his head to the normal position. As the shoulders are slowly lowered the head falls back in advance of them, the occiput striking the bed quite forcibly. This sign has occasionally been found when most others have been indefinite or absent. Perfect lucidity must accompany this sign for it to be of value. It will, of course, always be present in an unconscious or stuporose patient. If the sign appears positive, it is wise to eliminate the element of inattention by saying: "Come on, old chap; lift your head up." The ease with which the normal child does it contrasts with the obvious effort of the child with paresed neck muscles.

Kernig's Sign.—Kernig's sign is usually present in some degree; but the normal variation is so marked that it is often difficult to assess its diagnostic value. A complaint of pain when the leg is more than 30° from the vertical is definitely abnormal.

The Abdomen.

In several cases the bladder has reached to the umbilicus.

Weakness of a Muscle Group.

Weakness of a group of muscles will not be discussed here beyond the statement that in addition to the various limb groups we have had respiratory involvement in an unusually high proportion of cases (33%). Two cases with facial weakness[1] have occurred, and one with unilateral hypoglossal and glosso-pharyngeal weakness.

The Cerebro-Spinal Fluid.

It is possible to do a lumbar puncture too early in the disease and obtain a normal fluid. This occurred in two of the cases. Also it rather tends to mask the spinal signs for the subsequent examinations. Cells have numbered up to 2,500 per cubic millimetre. In the early stages there have been many more polymorphonuclear cells than usual. Lymphocytes alone have been present later.

The Diagnosis.

It is obviously impossible to discuss all the myriad clinical conditions which may simulate poliomyelitis in its early stages—and by early I mean in the first twenty-four hours; for that is the stage we are seeing them in now. One may mention the various types of meningitis, meningismus, the influenzas, rheumatism, the bowel toxæmias, catarrhal jaundice, all the upper respiratory infections, stiff necks, painful limbs from trauma, the exanthemata and acute rheumatism, and still leave many loopholes.

It is, of course, much easier to give a positive diagnosis than a negative one. It has been our experience that when poliomyelitis is present, it is present alone. The presence of any unusual symptoms, such as earache, cough, rapid respiration or diarrhœa, makes one very guarded. The peculiar features of the signs as indicated are our diagnostic yardsticks; of course, many abortive types will occur and remain forever undiagnosed.

Summary of Points in Diagnosis.

The following points are regarded as the most helpful in diagnosis and differential diagnosis:

1. The symptom volunteered (without suggestion from the parent) of pain in the back and/or back of the neck.
2. The parent's observation of drowsiness, which is not present when the doctor is examining the child, and an observation by the parents of "twitchings" "tremblings" or "the shakes".
3. Anorexia, which is practically constant (a good appetite is rather strongly against the diagnosis).
4. Some difficulty in commencing the act of micturition, or the finding of a bladder up to the umbilicus.
5. The presence of neck stiffness (without head retraction), spine stiffness, Amoss's sign or a head-

[1] Up to the time of publication many more cases of facial paresis have occurred, some so gross as to resemble Bell's palsy.

lag in a perfectly lucid and cooperative child with a temperature of 37·8° to 39·5° C. (100° to 103° F.).

6. The child's age, which has been predominantly from three to ten years.

Conclusion.

May I conclude on a somewhat depressing note? I feel that once a child contracts the disease its progress in the acute phase is beyond our control at present. We can nurse the children, see to their backs and skin, provide them with respirators and intravenous therapy if they need it, and give them the inestimable benefit of early correct splinting so strongly emphasized by Dr. Jean Macnamara; but the limiting of the destructive action of the virus in those delicate and highly specialized motor cells, I feel, is at the moment utterly beyond our powers. It may or may not be beyond that of the defensive mechanism of the child; the progress of the case is the only answer we have to that all-important question.

Reports of Cases.

The following are typical case histories, taken in the patients' homes.

CASE I.—J.B., a girl, aged six years, had been sick for one week. Seven days previously she had had a sickly turn, when she felt "sick in the stomach" and had a headache. The mother did not think she was feverish then, and there was no vomiting. Next day she had been "a little grizzly and off her food". The following day, that is, four days before I saw her, she still complained of headache and felt sick. Three and two days before I saw her she seemed much better, but was still irritable, not drowsy. The day prior to coming under observation she felt much better, till the afternoon, when she again complained of headache, and vomited for the first time. She seemed to be feverish in the night. At 4 a.m. on the day I saw her she cried because of pain in the back, saying: "Oh, my back's hurting, oh, my back's hurting." She was drowsy and would sleep if left quietly. When her mother sat her up for a drink today, she said: "Oh, my back." She had no cough or sore throat. Previous illnesses included measles, mumps and pneumonia.

On examination the child was found to be flushed, lucid and cooperative. The temperature was 38·6° C. (101·5° F.) and the pulse rate 140 per minute. Respiration was easy and quiet. The tongue was heavily coated; the throat was normal; there was no nystagmus. There was some indefinite neck stiffness. She was unable readily to look at her umbilicus and could not kiss her knees (she could do this easily the previous night), and she complained of pain in the back when she attempted it. Kernig's sign was present. There was slight tremor, equal in both arms. There was no paresis. A diagnosis of probable poliomyelitis of the dromedary type was made.

Later it was found necessary to put this child in a respirator.

CASE II.—B.D., aged seven years, had been well until 4 a.m. on the previous day, when he had called his parents because of headache and feeling sick. He stayed in bed all that day, and about midday said: "My neck is sore." He was feverish. There was no complaint of pain in the back. He was slightly drowsy. The appetite was poor. There was some stiffness of the neck, and still frontal headache. The patient's nose had bled on the day I saw him, and he had a slightly sore throat. He was very thirsty. There were no pains in the limbs. He had complained of seeing double that afternoon.

The temperature was 39·2° C. (102·7° F.) and the pulse rate was 104 per minute. The face was flushed. The child's mind was clear and he answered questions excellently. His nose was bleeding. There was some general reddening of the throat, but no exudate. There was marked stiffness of the neck and spine. Amoss's sign was absent. Head lag was very pronounced. There was evidence of a slight right facial weakness.

A diagnosis of poliomyelitis of an acute type was made.

The third case is a very good example of the "difficult" case.

CASE III.—J.M., a male, aged seven years, had felt quite well till the morning of the day on which I saw him first. He had wakened complaining of violent pains in the head (frontal). He had breakfast and his headache had become more severe later. He went to bed and slept. He had no teichopsia. He ate his dinner, but vomited after it. That afternoon he seemed drowsy and feverish, and had some abdominal pains. He did not complain of pain in the neck or back except while being examined. He cried at one stage on account of headache. There was no evidence of intestinal or urinary disturbance. He had never had measles.

The temperature was 38·2° C. (100·7° F.), the pulse rate was 90 and respiration rate 20 per minute. The child answered questions normally. The throat was reddened and engorged. There was a very slight suggestion of stiffness of the neck on full flexion. On looking at the navel, he was not able to bend his head perfectly freely; but the limitation was very slight as yet. He was able to kiss his knees. Amoss's sign and head lag were absent. There was no paresis. Kernig's sign was absent.

The case was regarded as too indefinite to label poliomyelitis as yet, though the story was suggestive and the epidemic had just spread to the town where the child had lived. The condition might be one of upper respiratory tract infection with some fibrositis only. It was considered justifiable to keep him under observation for 24 or 48 hours.

A later report showed that the condition cleared up completely in a day or so. He may have had abortive poliomyelitis.

THE FUNCTIONAL PATHOLOGY OF ANÆMIA.
IV: THE SYMPTOMS AND SIGNS OF ANÆMIA.[1]

By C. G. LAMBIE.
(From the Department of Medicine, University of Sydney.)

SYMPTOMS.

Introduction.

THERE is a general similarity between the symptoms of anæmia and those of disorders of the circulation. This has frequently been explained by ascribing both to anoxæmia. A closer scrutiny reveals the fact that the similarity is not so great as might appear from a mere enumeration of the symptoms. The resemblance is more marked in the case of certain varieties of circulatory failure than in others; in these forms, as we shall see, there may be a common underlying factor.

Taking the commonest type of failure of the circulation, namely, congestive failure, and comparing the cardinal symptoms with those of anæmia, the most notable difference between the two has reference to the order of development of the symptoms and the relative prominence of the complaints in the patient's consciousness. If the patient with congestive failure is asked: "What is it that troubles you most?" he will probably reply:

[1] Parts I, II and III of this paper were published in the issues of August 21 and 28 and September 4, 1937, respectively. A *résumé* of the four parts of Professor Lambie's paper was read at a meeting of the Section of Medicine of the New South Wales Branch of the British Medical Association on April 22, 1937.

"Shortness of breath." If the same question is put to the anæmic patient, his answer will usually be: "Weakness, tiredness or lassitude." Again, if the patients were questioned regarding the chronological order of development of the symptoms, the patient with congestive failure would give dyspnœa as the first symptom and weakness as a late symptom, whereas the anæmic patient would give weakness as an early and dyspnœa as a late symptom. The main symptoms may therefore be arranged in the following approximate order in point of both time and severity.

Congestive Failure.	*Anæmia.*
Dyspnœa.	Weakness.
Giddiness, faintness, nausea.	Palpitation.
Consciousness of the heart's action.	Giddiness, faintness, nausea.
Weakness.	Dyspnœa.

The explanation of these differences lies in the fact that whereas the symptoms of anæmia are those of gradually developing oxygen lack, the most prominent symptom of congestive failure, dyspnœa, is not due to oxygen lack. In congestive failure the patient may complain of dyspnœa, although the cardiac output[20][24] and the arterial oxygen saturation[23][51] may be normal; therefore, it is not a question of oxygen lack. The dyspnœa in this condition is due to reflex stimulation of respiration by impulses arising in the congested lungs[34] and the mechanical embarrassment of respiration by diminished vital capacity.[59] It is true that congestive failure may ultimately be complicated by diminished cardiac output or by inability of the heart to respond to exercise, with consequent impairment of oxygen supply to the tissues. When this happens, the symptoms of oxygen lack—giddiness, faintness, nausea and weakness—appear and the dyspnœa is aggravated. Palpitation, or rather consciousness of the heart's action, in congestive failure is commonly due to the presence of cardiac irregularities (such as auricular fibrillation) or to hypertrophy of the left ventricle, and its order of appearance varies.

In anæmia true palpitation is a prominent symptom, and other cardiac irregularities are uncommon. In anæmia the dyspnœa has little, if anything, to do with pulmonary reflexes. The vital capacity is normal[60] or only slightly diminished.[73] According to Peabody and Sturgis,[61] weakness is not a factor in causing diminished vital capacity. The slight diminution may perhaps be due to active hyperæmia of the lungs. In those types of failure of the circulation associated with diminished cardiac output (as in oligæmic peripheral failure), weakness, faintness and palpitation are prominent symptoms, and, as in anæmia, they are to be ascribed, partly at all events, to oxygen lack.

In the last resort, the symptoms of anæmia, like those of mountain sickness, are due less to insufficient oxygen supply to the body as a whole than to deficient oxygen supply to the brain. The diversion of blood supply to other organs from any cause, physical or psychical, may precipitate symptoms.[4]

Weakness and Lassitude.

The symptoms of weakness and lassitude are very similar to those observed in mountain sickness.[3][4][16][40][68] The patient not only complains of disinclination for effort, but a greater effort is necessary in order to carry out muscular movement; moreover, effort produces rapid tiredness. This is not the tiredness experienced after prolonged muscular exercise, but rather a heaviness and lassitude which disappear after a short rest.

The fatigue of muscular exercise is largely peripheral in origin and associated with the development of lactic acid acidosis in muscle,[22][40][53] with a resulting paresis of nerve endings. The weakness in anæmia is evidently of central origin and is probably due to the presence of a low oxygen tension in the central nervous system. It is true that the diminished oxygen supply to muscles might be expected to cause the rapid development of lactic acid acidosis and peripheral fatigue. It has also been shown that the mechanical efficiency of the muscles is impaired in anæmia and in anoxæmia.[78] No data seem available, however, regarding the lactic acid in the blood during muscular effort in anæmia in man. This may be due to the difficulty of pushing voluntary muscular effort to the point at which peripheral fatigue develops. Fatigue of central origin is evidently the most important limiting factor in muscular effort in anæmia. The term "exhaustion" should be applied to a combination of central and peripheral fatigue. It is usually accompanied by tremulousness, as well as by fatigue and paralysis of movement. Any considerable effort in anæmia would rapidly give rise to exhaustion.

Palpitation.

As the output of the heart may be considerably increased in anæmia, it would at first seem reasonable to attribute the palpitation to the increased cardiac activity. As we have seen, however, the cardiac output is not greatly increased until the hæmoglobin falls below 50%, and even then the increase is achieved chiefly by means of greater stroke volume rather than by acceleration; but palpitation may be observed with much lesser degrees of anæmia. Palpitation is also observed at high altitudes before the oxygen tension in the atmosphere has fallen to the point at which increased cardiac output occurs.[3] Both in anæmia and at high altitudes, even when the basal pulse rate is normal, less exercise is necessary to produce a given acceleration than in the normal individual at sea-level.[3][41] It therefore appears that the cardio-accelerator centre is in a more sensitive condition, owing probably to the low oxygen tension in the central nervous system. Any stimulation of the centre, whether by impulses from the higher centres or through reflex channels, would increase its metabolism, thereby aggravating the condition of local oxygen lack and causing a rise in carbon dioxide tension, to both of which it would react by increased rapidity of discharge. What happens in regard to the cardio-inhibitory centre is not clear, but a diminution in vagus tone might also be caused

by the local oxygen lack and carbon dioxide excess. Such stimulation of the centres would occur as a result of cortical overflow during voluntary muscular effort, and also as a result of stimuli reaching them from the centres concerned with emotion. Muscular effort would also affect the centre reflexly, particularly through the Bainbridge reflex, which would be excited by increased venous return. Muscular effort, emotion and reflex disturbances caused by such things as abdominal distension or pain are well known to induce palpitation, especially in anæmic subjects.

Palpitation outlasts the stimulus producing it, probably because it fails to increase the cardiac output and to restore the normal gaseous tension in the centres; also because the symptom itself causes distress and further emotional disturbance. Why palpitation should occur when the heart is accelerated from some causes and not from others is not quite clear. It seems as though the sensation is produced most readily when acceleration and augmentation are much out of proportion to the diastolic filling of the chambers.

Palpitation is one of the factors which limit effort in anæmia. The discomfort in the thorax and the "suffused" feeling at the root of the neck are often confused with dyspnœa, and careful cross-examination may be necessary to distinguish between them.

Giddiness, Faintness and Syncope Tinnitus.

Giddiness, faintness, syncope and tinnitus are met with not only in anæmia, but in oxygen lack from other causes. In anæmia the brain would seem to be able to tolerate a lower oxygen tension than that which would cause symptoms in acute oxygen lack, but the margin between the actual oxygen tension and that which would cause symptoms is probably narrowed. Unfortunately no observations have been made on the correlation between symptoms and the oxygen tension in venous blood leaving the brain, such as have been reported in other conditions.[46] As the brain is largely dependent for its oxygen supply upon a compensatory increase in blood flow, any diminution in blood flow would rapidly induce the symptoms of oxygen lack. Such a diminution in blood flow could be brought about by a diversion of blood to other parts of the body or by diminished cardiac output. As the pressure reflexes even at rest have to function actively in order to maintain the blood pressure in the presence of the diminished blood viscosity and capillary dilatation in muscles, they may fail to compensate adequately for change of posture or for the further capillary dilatation accompanying muscular effort. Therefore the assumption of the erect posture or the performance of muscular effort may induce giddiness or even syncope. Similarly, emotion, by causing vasodilatation in the skin or splanchnic area and tachycardia, may cause the same symptoms. Palpitation is not infrequently followed by giddiness or faintness, partly because the tachycardia may cause diminished cardiac output, and partly because it induces emotional excitement. It is uncertain whether the giddiness is due to oxygen lack in the labyrinths or of the cerebral centres concerned with equilibration. A similar uncertainty applies to tinnitus in relation to the auditory apparatus.

Lack of Concentration, Insomnia, Nightmare, Delusions, Depression or Excitement and other Mental Aberrations.

Lack of concentration, insomnia, nightmare, delusions, depression or excitement and other mental aberrations can all be attributed to oxygen lack of the higher centres.

In diseases such as pernicious anæmia and pellagra, in which degenerative changes in the central nervous system accompany the anæmia, the mental symptoms may bear no direct relationship to the degree of anæmia. Gulland and Goodall[82] cite a case of pernicious anæmia in which the mental symptoms became worse as the blood improved, and *vice versa*.

Hyperpnœa and Dyspnœa.

As already explained, hyperpnœa on effort is probably to be ascribed to the effect of cortical overflow or reflex stimulation upon the sensitized respiratory centre. It is difficult to see how oxygen lack could be the cause, as oxygen lack merely depresses the centre[24][25][70][72][77] and muscular effort would not affect the oxygen supply to the chemo-receptors of the carotid body so long as the arterial oxygen saturation remained normal and the blood flow through the carotid bodies was maintained. On the other hand, if carbon dioxide, which would be produced in increased amounts in the stimulated centre, was not effectively removed by the anæmic blood, it would stimulate the centre directly. The pressure reflex of the carotid sinus may also be to some extent involved in stimulating the respiration during exercise, as the effect of cortical overflow upon the vasomotor centre may not cause sufficient arteriolar constriction to balance capillary dilatation, so that compensation through the carotid sinus would become necessary. A fall in pressure in the region of the sinus stimulates the respiratory[35][37] as well as the vasomotor centre.

Dyspnœa, which is a subjective sensation, has to be distinguished from hyperpnœa, which is an objective sign. In dyspnœa afferent stimuli from the organs of respiration are important, while hyperpnœa may occur without the unpleasant sensation which we call dyspnœa. Dyspnœa occurs most readily when there is some obstruction to respiration, or some embarrassment of the respiratory movements with diminution in the vital capacity. In anæmia these are usually lacking, and in many cases of anæmia respiratory distress is so little pronounced that weakness and palpitation, rather than dyspnœa, are the limiting factors in effort. In order to produce dyspnœa in the absence of diminished vital capacity or obstruction, it is necessary as a rule to produce a very marked degree of hyperpnœa.[74] If, for example, carbon dioxide inhalation is used to stimulate the breathing in a normal person, the tidal volume has to be increased to two-thirds of the vital capacity or more before

any distress is experienced. In the anæmic patient, however, respiratory distress appears to be produced with lesser degrees of hyperpnœa than this, but exact observations are required in order to confirm this impression. The probable explanation is that oxygen lack causes peripheral fatigue in the muscles of respiration, and this, combined with central fatigue, gives rise to exhaustion. This is especially pronounced if the extraordinary muscles of respiration are brought into play. It is remarkable, however, how patients at rest come to tolerate hyperpnœa, when it is of long standing, without experiencing much distress, while a similar grade of hyperpnœa produced suddenly by muscular effort is accompanied by dyspnœa. The dulling of the senses by oxygen lack may ultimately diminish the distress, but the retention of carbon dioxide by increasing the dissociation of oxygen from hæmoglobin would act in the opposite direction. Dyspnœa at rest occurs only in the terminal stages of anæmia, and it may last only for a brief period. Dyspnœa may, of course, be due to other causes in diseases associated with anæmia, and this applies especially to thoracic or abdominal tumours or to accumulations of fluid which embarrass the respiratory movements and reduce the vital capacity.

Headache, Nausea and Anorexia.

Headache, nausea and anorexia are all common symptoms of oxygen lack and are observed at high altitudes[3] as well as in anæmia. How far headache is due directly to oxygen lack or indirectly to cerebral œdema, due to increased permeability of the cerebral capillaries,[45] is unknown.

Pain.

As oxygen lack is believed to be a factor in the genesis of cardiac pain and of pain in voluntary muscles, it is noteworthy that pain is not a prominent feature of anæmia, in spite of the presence of oxygen lack. The conditions are, however, unlike those present in ischæmia, for the blood flow in anæmia is free and even increased, so that the metabolites which are responsible for exciting pain[47] would be rapidly removed and would not attain a sufficient concentration in the tissues. If, however, anæmia is added to ischæmia, due to arterial disease, pain is more readily produced than when the blood is normal, and cure of anæmia may abolish the pain.[14][33][36][56] It has, however, been reported that pain can be produced in the absence of vascular disease and may even occasionally be a limiting factor in effort.[21][64] Without denying such a possibility, the evidence must be regarded with a certain amount of reserve, as most of the patients studied have been past middle age or over the age of thirty, and disease of the smaller vessels may be difficult to detect at the bedside or even with the naked eye *post mortem*.

Signs.

Pallor (Cyanosis).

The principal cause of the pallor is obviously the diminished hæmoglobin percentage. In addition, the emptying of the skin vessels owing to the diversion of blood elsewhere may play a part. Cyanosis is not a feature of anæmia, in spite of the increased desaturation of blood in the capillaries. Cyanosis, as Lundsgaard and Van Slyke[49] have pointed out, is not dependent upon the relative proportions of reduced hæmoglobin and oxyhæmoglobin, but upon the total amount of reduced hæmoglobin in the blood. It requires at least five grammes of reduced hæmoglobin per 100 cubic centimetres of blood to produce cyanosis. Hence cyanosis cannot occur if the hæmoglobin content of the blood is less than five grammes per 100 cubic centimetres, which is equivalent to 30% of the normal hæmoglobin value.

Empty Veins.

In marked anæmia the superficial veins of the arm may become less prominent, probably owing to the constriction of veins which has already been referred to.

Acceleration of the Pulse.

A slight but variable increase in the pulse rate may be observed when the hæmoglobin falls to 60%.[5] (See Figure XXIX.) With hæmoglobin values below 50% some increase in pulse rate is common, but as a rule it is not very great, as the increased cardiac output is achieved chiefly by increased stroke volume. The increase is probably due partly to the effect of oxygen lack or carbon dioxide upon the medullary centres, and partly to the excitation of the Bainbridge reflex by increased venous return; also to stimulation of the cardio-accelerator mechanism through the carotid sinus reflex, on account of diminished viscosity.

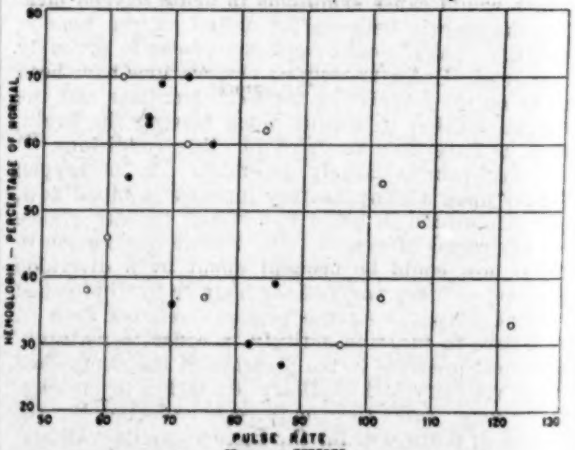


FIGURE XXIX.

Chart showing relation between hæmoglobin concentration (percentage of normal) and pulse rate in patients with "primary" anæmia (solid dots) and anæmia secondary to diseases other than carcinoma (circles). [After Blumgart, Gargill and Gilligan (*Journal of Clinical Investigation*, Volume IX, 1930).]

While the basal pulse rate may be almost normal, a greater amount of acceleration occurs for a given amount of exercise than in the non-anæmic state, for reasons already explained in the discussion on palpitation.

A terminal asphyxial slowing of the pulse is occasionally observed.

Fall in Blood Pressure.

Acute oxygen lack caused by breathing atmospheres deficient in oxygen produces a slight transient rise in blood pressure.[71] In anæmia no rise of blood pressure is observed. As a rule the blood pressure remains about normal with hæmoglobin values above 50%; but below this the blood pressure is frequently diminished, the maximum pressure often falling to about 100 millimetres of mercury. This fall is probably due to failure of arteriolar constriction and of cardiac output to compensate for capillary dilatation and diminished viscosity. In long-standing cases of anæmia, degenerative changes in the heart muscle may impair the ability of that organ to increase its output.

Cardiac Dilatation and Hypertrophy.

Some degree of cardiac dilatation is frequently met with,[2][9] but as a rule it is not marked, the left border of the deep cardiac dulness being seldom much outside the mid-clavicular line in cases uncomplicated by vascular disease. The possible causes of dilatation are loss of tone, due to oxygen lack, and fatty changes in the heart muscle on the one hand, and increased work of the heart on the other. There is seldom much hypertrophy, which is perhaps surprising in view of the work the heart may be called upon to perform even at rest, and the absence of hypertrophy has been used as an argument against the view that a compensatory increase in cardiac output occurs. It may be, however, that the importance of the load on the heart has been exaggerated. There is no marked increase in cardiac output until the hæmoglobin falls below 40% to 50%. With a hæmoglobin value of 30% and a cardiac output of eight litres per minute the heart would be doing at rest an amount of work no more than that which would be required for moderate exercise; and in estimating the work performed it is necessary to allow for the fact that the blood pressure may be diminished and so lighten the work on the heart. It is only when the hæmoglobin falls to between 20% and 30% that the cardiac output may be such as might be expected to throw a great strain on the heart. The question then arises as to how long such a condition would have to persist in order to produce much hypertrophy. An anæmia of this degree might be expected, in the majority of cases, to terminate before long with the death of the patient, or in recovery. The degenerative changes in the heart muscle and the impaired oxygen supply may also militate against the development of hypertrophy.

Cardiac and Vascular Murmurs.

Cardiac hæmic murmurs have usually been explained on the analogy with the mechanism of murmurs due to structural changes in the heart and vessels. Thus they have been attributed to dilatation of the vessels beyond the pulmonary and aortic orifices or to "relative" incompetence of the mitral and tricuspid orifices, variously ascribed to dilatation of the ventricles, relaxation of the auriculoventricular muscular rings or weakness of the papillary muscles leading to imperfect closure of the valves.

Balfour[1] accounted for the localization of the basal murmur to the left of the pulmonary area by supposing that mitral regurgitation set up vibrations in the left auricular appendix.

It must be admitted, however, that there is little, if any, evidence to support these speculations. No dilatation of the pulmonary artery or of the aorta has been demonstrated radiographically or at *post mortem* examination; and as for dilatation of the ventricle, hæmic murmurs may be present towards the mitral area in the presence of little demonstrable dilatation, while murmurs may be absent in spite of definite dilatation. More precise data (for example, that yielded by X ray examination), correlating the appearance of hæmic murmurs with variations in the dimensions of the heart and vessels, are required. If the mitral orifice were really incompetent, whether this incompetence was referred to as "relative" or otherwise, some of the other phenomena which commonly accompany incompetence might be expected to be in evidence—diminished vital capacity, accentuation of the second pulmonary sound, dilatation or hypertrophy of the right ventricle—but no correlation between systolic mitral hæmic murmurs and any of these has been established. Incompetence, if it occurs, must be very slight and very perfectly compensated by the left auricle and by the sphincteric action of the muscles guarding the orifices of the pulmonary veins.

It is a well-established fact that murmurs can be produced when blood or other fluid passes from a narrow orifice into a wider tube (Corrigan,[15] 1829, Chauveau,[13] 1858), and that it is not the friction between the blood and the walls of the vessels, as supposed by Gendrin[26] (1891), but the eddies or "fluid veins" (Savart,[69] 1833) set up within the fluid (Heynsius,[38][39] 1854, 1878) which are the cause of the vibrations that are transmitted to the vessel walls and ultimately to the ear. It is also true that these eddies would be set up more readily the less viscous the fluid and the greater the velocity of flow. If the appearance of murmurs could be definitely correlated with these two factors—diminished viscosity and increased blood flow—both of which are present in anæmia, we should feel on firmer ground; but adequate data are lacking. It might be then possible to account for some murmurs without postulating any anatomical changes in the heart or vessels. For example, if it is a fact that the internal jugular vein changes its calibre as it leaves the cervical fascia and debouches into the innominate vein, the humming top murmur, or *bruit de diable*, could be accounted for without it being necessary to suppose that there is any abnormal constriction of the vein from without by the cervical fascia in anæmia. If the cervical fascia and muscles of the neck are put on the stretch, as by turning the head to one side, the murmur, if present, is aggravated, as is to be anticipated, owing to further narrowing of the jugular vein by compression from

without; but this is no evidence that the narrowing is any greater than normal, because the same degree of narrowing which would produce a murmur when the blood is anæmic, may not suffice to cause a murmur if the blood is normal. The conditions would be particularly favourable to the production of the murmur in diseases such as chlorosis, in which the blood volume (and consequently the cardiac output) is much increased and the corpuscular volume (and therefore the viscosity) is low.

In connexion with the mechanism and relative incidence of murmurs at the basal orifices, the work of Blackhall-Morison[53] on the little muscles of the heart is very suggestive. He has drawn attention to the detrusive action of the muscles of the *conus arteriosus* upon the pulmonary orifice. The narrowing of the exit of the ventricle caused by the contraction of these muscles during ventricular systole might give rise to a sufficient disparity in size between the outlet of the right ventricle and the pulmonary artery to give rise to a murmur in the presence of anæmia, although not under normal conditions. Again, if the muscular supports of the pulmonary valve were to yield, a functional diastolic murmur might result.

The conditions at the aortic orifice are somewhat different, the muscles connected with this structure being more concerned with rotation of the base of the aorta in the direction of the general ventricular twist in systole. The valve does, however, receive a certain amount of support from the small muscles, especially during diastole. Goldstein and Boas[27] (1927) report the occurrence of aortic diastolic murmurs in anæmia unaccompanied by any evidence of valvular or aortic disease at *post mortem* examination. They attribute them to relaxation of the aortic ring, resulting in functional incompetence. This condition might perhaps be due to loss of tone of the small muscles referred to.

It is not easy to suggest a satisfactory explanation of the systolic murmurs heard towards the apex of the heart. There has even been some doubt as to whether they are endocardial. The persistent contraction of the musculature of the lower auricular segment has a sphincteric action during ventricular systole.[57][58] Again, contraction of the papillary muscles and of specialized ventricular muscle at the base of the auriculoventricular valve segments serves to close the valves efficiently.[53] It would therefore be a very attractive hypothesis to ascribe the systolic murmurs to loss of tone of these muscles, with resulting regurgitation. If the loss of tone of these specialized structures could occur with little, if any, general dilatation of the heart, one objection to the relative incompetence theory would be removed; but this supposes that oxygen lack or toxæmia would have a selective action on the tone of these specialized muscles, since a general loss of tone would cause dilatation. As already pointed out, the chief difficulty in accepting this explanation is the absence of any other evidence of regurgitation.

Another possibility is that the murmurs may have something to do with the whirling and vortiginous movement of blood within the ventricle becoming exaggerated by the same factors as those previously mentioned, namely, diminished viscosity and increased blood flow. Against this hypothesis is the occasional persistence of the murmur after the hæmoglobin has returned almost to normal. This suggests that whereas venous murmurs and systolic murmurs at the base may be truly "hæmic" (that is, due to physical changes in the blood), those at the apex may be functional (that is, due to altered function of the cardiac muscle), but it would be useless to speculate further in view of the many gaps and uncertainties in our knowledge.

Cardiac Irregularities.

The rarity of cardiac irregularities, in spite of oxygen lack and degenerative changes in the heart muscle, has already been noted in the discussion on failure. The free coronary circulation and rapid removal of metabolites might have something to do with this. Occasionally some flattening of the *T* wave and heart block are observed in advanced cases; otherwise, apart from showing acceleration, the electrocardiogram remains normal (Reid[65]).

Albuminuria.

The urine in anæmia is usually normal; but in severe anæmia albuminuria may be present. Evidently the blood flow through the kidneys must usually be adequate to prevent the development of anoxic albuminuria.

Œdema.

As Landis[45] has shown, oxygen lack increases the permeability of capillaries. It may also cause the accumulation of metabolites in the tissues, increasing their affinity for water. The very low tissue oxygen tension which occurs in advanced anæmia is probably the most important cause of œdema. Other causes may, however, play a part, but less is known regarding them. Terminal failure of the circulation, for example, might be partly responsible. It would, of course, increase the oxygen lack; but before it can be decided whether venous congestion or peripheral failure are factors of importance, more information will have to be forthcoming regarding the venous pressure and the difference in corpuscular volume between the arterial and the venous blood. More information is also required regarding another possible cause of the œdema—diminution in colloid osmotic pressure from changes in the plasma proteins. A diminution in fibrinogen and in the total serum proteins, especially the albumin fraction, has been reported in some anæmias.[31][42][43][49][52][62][67] Finally the œdema might be nutritional in origin, owing to insufficient intake of protein. This might occur when both the anæmia and the œdema are primarily due to dietetic deficiency, or in cases in which, owing to illness, there is difficulty in getting the patient to take an adequate diet. What rôle,

if any, toxins associated with diseases causing anæmia play in the production of anæmia is unknown.

Digestive Disturbances.

Apart from anorexia and nausea or even vomiting, any of which may result from oxygen lack, digestive disturbances do not figure prominently in anæmia. Digestive disorders are more commonly due to the disease causing the anæmia; for example, gastric ulcers with or without vitamin deficiency, changes in gastric secretion in pernicious anæmia and neoplasms of the alimentary tract, all of which may cause anæmia, also cause local disturbances of function. The same applies to disease elsewhere which, either reflexly or through the production of toxins or metabolic disorders, may cause alimentary symptoms as well as anæmia.

Amenorrhœa and other Disturbances of Menstrual Function.

Here again it is very doubtful whether the disturbance of menstruation is attributable to the anæmia as such. Amenorrhœa is a common but not an invariable accompaniment of anæmia. On the other hand, even slight degrees of anæmia associated with certain diseases, such as pulmonary tuberculosis, are prone to be accompanied by amenorrhœa. This suggests that toxic, nutritional and endocrine disturbances or local diseases of the reproductive organs may be the common cause of the anæmia and of the amenorrhœa. That oxygen lack may disturb the functions of the ovary and other endocrine glands controlling menstruation, as well as the uterus itself, is, of course, a possibility, but more evidence is required in support of it.

Rise of Temperature.

Rises of temperature are frequently met with in patients with anæmia, and they are usually attributed to infection, toxæmia or, as in leuchæmia and Hodgkin's disease, to metabolic disturbances peculiar to the disease in question. These are undoubtedly among the causative factors in a large number of cases of anæmia, but it may be doubted whether they account for pyrexia in all cases. Some elevation of temperature is very common in anæmia, arising from a great variety of causes. On the analogy with what occurs in muscular exercise, in which the heat loss by the skin may be insufficient to balance that produced in the muscles owing to the shift of blood to the internal organs, it is just possible that a similar shift of blood in anæmia may impair the heat loss from the skin; and this, together with the increased metabolism due to the work of the heart and muscles of respiration, may so disturb the balance between heat production and heat loss as to cause an elevation of temperature. In support of this possibility one may cite the observation of Tompkins, Brittingham and Drinker[76] that transfusion of blood to patients with pernicious anæmia was followed within twelve hours by a return of the temperature to normal if it had previously been elevated. That the return to normal was not immediate may be accounted for, first by the fact that it takes some time for capillaries (for example, those of muscle) to recover their normal tone after prolonged exposure to oxygen lack, so that the blood shift might persist for some time; and secondly by the continued elevation of the metabolic rate, owing to the work of the heart remaining greater than normal so long as the organ had to deal with the increased blood volume resulting from the injection of fluid into the circulation.

Altered Metabolic Rate.

The experiments of Tompkins, Brittingham and Drinker,[76] quoted above, afford the best evidence of some stimulating action of anæmia upon metabolism. Abolition of the anæmia by means of blood transfusion caused the metabolic rate to return to normal in a day or two. This elevation of metabolism is paralleled by the rise in metabolism which occurs during a certain phase of anoxæmia produced by breathing atmospheres deficient in oxygen, and probably has a similar cause, namely, increased work of the heart and muscles of respiration. On the other hand, there is reason to believe that the metabolism in the tissues would be diminished by oxygen lack of a sufficient degree; and this may eventually outbalance the effect of increased cardiac and respiratory activity, so that a depression of the total metabolism would result. It is to be expected, therefore, that the metabolism rate would alter at different stages of anæmia, depending upon a balance of factors, some making for increase and others for decrease in metabolism. Besides oxygen lack, numerous other factors may influence the respiratory metabolism in disease causing anæmia, some tending to increase, others to diminish it. The variable results obtained by different observers[12][17][18] and the difficulty of drawing conclusions are therefore not surprising. Not only is there a lack of agreement from case to case, but also between observations upon the same patient at different times. The correlation between metabolic rate on the one hand and blood picture, treatment, the presence of pyrexia and respiratory embarrassment on the other has also been insufficiently noted. A mere statistical summary of the metabolic rates found in different kinds of anæmia, such as that shown in Table II, gives an inadequate conception of the factors involved.

A few general facts, however, seem to emerge from these results. Of all diseases of the blood, the leuchæmias are those associated with the most marked and constant elevation of metabolism. This is not entirely related to the degree of anæmia or to the pyrexia which is frequently present, but appears to be due to a specific stimulus peculiar to the disease, possibly associated with the metabolism of nucleo-proteins. In most anæmias the metabolic rate is within normal limits. It is more frequently elevated in pernicious anæmia than in secondary or hypochromic and microcytic anæmias, while in very severe anæmia of any kind it may be diminished.

TABLE II.[1]

Composite Table showing Basal Metabolism in Anæmia and Leuchæmia.

	Cases.	Below −20%.	−20% to −16%.	−15% to −11%.	−10% to +10%.	+11% to +15%.	+16% to +20%.	Over +20%.
Secondary anæmia	30	—	3·3	—	80	13·4	—	3·3
Splenic anæmia and pernicious anæm. ..	36	2	—	3	53	8·0	19	14·0
Leuchæmia, lymphatic and myelogenous ..	15	—	—	—	27	7·0	6	60·0

[1] From Boothby and Sandiford's tables in *Journal of Biological Chemistry*, Volume LIX, 1922, page 783,[71] and *Physiological Reviews*, Volume IV, 924, page 69.[68]

Grafe[29][30] and Eberstadt[19] thought that the activity of the bone marrow was an important factor in elevating the metabolism. While it is difficult to disprove this possibility, Rolly[66] could not confirm Grafe's results. What consumption of energy is involved in the rapid growth of bone marrow cells is uncertain.

In assessing the factors influencing metabolism in diseases causing anæmia, two very common sources of fallacy are the neglect to take into account variation in the body temperature, and the increased work of the respiratory muscles which may result from embarrassment of respiration by the presence of abdominal or thoracic tumours (enlarged spleen, neoplasms, glandular enlargements) or accumulation of fluid. Among the factors making for diminished metabolism are the sedentary life of anæmic patients, diminished metabolism of tissues due to fatty change and replacement of active tissue by fat and water, possibly tolerance leading to diminished demand for oxygen, lesions of the nervous system causing paralysis and atrophy, general emaciation or atrophy, and finally anaerobiasis due to very low oxygen tension. The actual metabolism in anæmia is therefore a balance between those things which increase and those which diminish it, and these may be summarized as follows:

A. Increasing metabolism:
- (1) Rise of temperature.
- (2) Action of toxins.
- (3) Mechanical embarrassment of the respiration.
- (4) Increased work of the heart and muscles of respiration, especially when the oxygen tension and hæmoglobin percentage fall below the critical threshold.
- (5) Increased activity of the bone marrow (?).
- (6) Specific effect associated with abnormal protein metabolism in leuchæmia.

B. Diminishing metabolism:
- (1) Low oxygen tension causing appearance of anaerobic areas.
- (2) Diminished demand for oxygen due to—
  - (*a*) Sedentary life.
  - (*b*) Lesions of the nervous system.
  - (*c*) Tolerance (?).
  - (*d*) Fatty changes and replacements of active tissue by fat and water.
  - (*e*) Emaciation and atrophy.

The results of various investigators, set out in Tables III and IV, should be viewed in the light of the above.

TABLE III.

Respiratory Metabolism in Anæmia. Summary of Results Obtained by Various Investigators.

Diminished.	Normal.	Increased.	Authors.
Mild cases of secondary anæmia, chlorosis (under iron).	Most cases of anæmia.	Pernicious anæmia, some cases of secondary anæmia.	Magnus-Levy.[140]
Chlorosis.	Secondary anæmia.		Thiele and Nehring.[71]
Very severe cases of pernicious anæmia.	Mild cases of pernicious anæmia.	Severer cases of pernicious anæmia.	Meyer and Du Bois.[144]
Rabbits with exhausted bone marrow after hæmorrhage or treatment with phenylhydrazine.	Rabbits rendered anæmic, but with active bone marrow.		Grafe[29] (1912). Eberstadt[19] (1912).
Severe pernicious anæmia with signs of marrow deficiency (? aplastic).	Pernicious anæmia during period of moderate regeneration.	Pernicious anæmia during regenerative crisis.	Grafe[29] (1915).
Secondary anæmic (parasitic).	Chlorosis Hæmorrhage } Upper limit.	Pernicious anæmia.	Rolly (recalculated by Meyer[144] and Du Bois[144] for Moeh's formula).
	Secondary anæmia (lead poisoning) } lower limit of normality.	Carcinoma with aplastic anæmia. Leuchæmia.	Pettenkofer and Voit.[68] Kraus and Chvostek.[142] Bohland.[6] Recalculated by Meyer and Du Bois for Moeh's formula.[144] Grafe[29] (1911).

TABLE IV.
Basal Metabolism in Anæmia. Summary of Results by Various Investigators.

Observer.	Disease.	Sex.	Age. Years.	Weight in Kilograms.	Temperature.	Pulse.	Respiration.	Hæmoglobin *per centum*.	Respiratory Quotient.	Calories per Square Metre per Hour. Meeh's Formula.	*Per centum* Above or Below Normal.	Remarks.
Grafe, E.	Lymphatic leuchæmia.	M.	50	66·0	36·2	100	36	..	0·700	68·06	+98	Very dyspnœic; restless at times.
Grafe, E.	Lymphatic leuchæmia.	M.	50	67·8	36·4	100	32	..	0·840	52·00	+50	
Grafe, E.	Lymphatic leuchæmia.	M.	50	68·5	36·5	90	34	..	0·831	48·66	+40	
Grafe, E.	Lymphatic leuchæmia.	M.	56	75·0	36·2	80	22	..	0·881	36·75	+6	
Grafe, E.	Lymphatic leuchæmia.	F.	65	48·2	36·0	80	20	..	0·898	44·45	+28	Asleep most of time.
Grafe, E.	Lymphatic leuchæmia.	F.	65	48·0	36·2	88	24	..	0·820	46·92	+35	Asleep most of time.
Grafe, E.	Lymphatic leuchæmia.	M.	49	63·8	37·2	82	22	..	0·851	65·04	+87	Restless now and then.
Grafe, E.	Lymphatic leuchæmia.	M.	56	65·8	36·6	88	—	..	0·807	49·83	+44	
Magnus-Levy.	Lymphatic leuchæmia.	M.	49	53·5	..	..	..	..	0·783	45·13	+30	Average 3 observations.
Grafe, E.	Myelocytic leuchæmia.	F.	32	64·3	36·6	82	20	..	0·783	53·20	+53	
Grafe, E.	Myelocytic leuchæmia.	F.	38	51·0	36·7	86	18	..	0·777	49·79	+43	
Grafe, E.	Myelocytic leuchæmia.	M.	48	57·5	36·5	80	15	..	0·814	42·33	+22	
Kraus, F.	Spleno - medullary leuchæmia.	M.	60	55·0	Normal.	86	12	50	0·790	48·67	+40	
Kraus, F.	Spleno - medullary leuchæmia.	M.	60	55·0	Normal.	86	12	50	0·772	46·40	+33	
Kraus, F.	Spleno - medullary leuchæmia.	M.	60	55·0	Normal.	86	12	50	0·777	45·83	+32	
Kraus, F.	Spleno - medullary leuchæmia.	M.	37	74·7	Normal.	100–108	14	45	0·811	47·80	+38	
Kraus, F.	Spleno - medullary leuchæmia.	M.	37	74·7	Normal.	100–108	14	45	0·814	46·72	+35	
Kraus, F.	Spleno - medullary leuchæmia.	M.	37	74·7	Normal.	100–108	15	45	0·805	49·37	+42	
Kraus, F.	Splenic leuchæmia.	F.	34	61·0	Normal.	80	14	45–50	0·866	49·74	+43	
Kraus, F.	Splenic leuchæmia.	F.	34	61·0	Normal.	80	12	45–50	0·808	49·17	+42	
Bohland.	Splenic leuchæmia.	F.	38	53·5	Normal.	114–120	22–24	—	0·800	51·84	+49	6 : 30 N.
Bohland.	Splenic leuchæmia.	F.	38	53·5	Normal.	114	22–26	—	0·890	57·80	+66	6 : 15 N.
Bohland	Splenic leuchæmia.	F.	38	53·5	Normal.	108	20–23	—	0·840	56·71	+63	6 : 45 N.
Bohland	Splenic leuchæmia.	F.	38	53·5	Normal.	112–116	19–23	—	0·810	54·80	+58	10 : 00 N.
Kraus, F.	Chlorosis.	F.	20	49·0	Normal.	78–84	16	45	0·710	41·17	+19	
Kraus, F.	Chlorosis.	F.	20	49·0	Normal.	78–84	18	45	0·735	44·29	+28	
Magnus-Levy.	Chlorosis.	F.	20	44·8	—	..	..	..	0·806	34·01	−0·2	13 observations.
Magnus-Levy.	Chlorosis.	F.	18	53·5	..	..	..	..	0·794	36·68	+7	8 observations.
Thiele and Nehring.	Chlorosis.	F.	22	57·5	Normal.	80	12	85	0·902	31·76	−8	Chronic *otitis media*.
Thiele and Nehring.	Chlorosis.	F.	22	57·5	Normal.	80	12	85	0·805	31·21	−10	Chronic *otitis media*.
Thiele and Nehring.	Chlorosis.	F.	22	57·5	Normal.	80	13	85	0·858	31·97	−8	Chronic *otitis media*.
Thiele and Nehring.	Chlorosis.	F.	18	58·5	Normal.	90–100	18	55	0·925	27·98	−19	
Thiele and Nehring.	Chlorosis.	F.	18	58·5	Normal.	90–100	18	55	0·800	27·89	−19	
Thiele and Nehring.	Chlorosis.	F.	18	58·5	Normal.	90–100	24	55	0·804	28·61	−17	Complained of toothache.
Thiele and Nehring.	Chlorosis.	F.	18	58·5	Normal.	90–100	24	55	0·779	29·63	−15	Complained of toothache.
Thiele and Nehring.	Chlorosis.	F.	18	59·0	Normal.	90–100	20	55	0·825	28·84	−17	
Thiele and Nehring.	Chlorosis.	F.	18	59·0	Normal.	90–100	20	55	0·806	29·03	−16	
Kraus, F.	Secondary anæmia.	M.	33	61·0	Normal.	88–100	13	25	0·752	40·73	+17	
Kraus, F.	Secondary anæmia.	M.	33	61·0	Normal.	88–100	14	25	0·763	39·48	+14	
Kraus, F.	Secondary anæmia.	M.	33	61·0	Normal.	88–100	14	25	0·746	43·99	+27	
Magnus-Levy.	Secondary anæmia.	F.	30	44·1	..	..	..	..	0·860	33·77	−3	2 observations.
Magnus-Levy.	Secondary anæmia.	M.	40	63·4	..	..	..	..	0·779	37·27	+7	4 observations.
Magnus-Levy.	Secondary anæmia.	F.	27	47·7	..	..	..	..	0·775	30·53	−12	8 observations.
Thiele and Nehring.	Secondary anæmia.	F.	—	47·5	Normal.	—	19	—	0·877	35·59	+3	Very anæmic after hæmorrhoid bleeding; slept part of time.
Thiele and Nehring.	Secondary anæmia.	F.	—	47·5	Normal.	—	18	—	0·850	34·53	−0·5	
Thiele and Nehring.	Secondary anæmia.	F.	—	47·5	Normal.	—	17	—	0·864	36·41	+5	Slept part of time.
Kraus, F.	Pernicious anæmia.	F.	46	54·0	Normal.	96–108	14	30	0·710	37·52	+8	
Kraus, F.	Pernicious anæmia.	F.	46	54·0	Normal.	96–108	15	30	0·709	40·55	+17	
Magnus-Levy.	Pernicious anæmia.	F.	40	44·7	...	..	..	..	0·813	38·87	+12	6 observations.
Magnus-Levy.	Pernicious anæmia.	F.	45	42·5	..	..	..	..	0·738	36·17	+4	1 observation.
Authors.	Pernicious anæmia, splenic.	M.	21	61·35	37·25	96	20	25	0·803	37·50	+8	
Authors.	Pernicious anæmia.	M.	27	52·72	37·25	100–124	24–28	20	0·839	42·99	+24	
Authors.	Pernicious anæmia.	M.	27	52·03	37·20	100–124	24–28	20	0·835	41·31	+19	
Authors.	Pernicious anæmia.	M.	32	47·80	38·00	100–124	20–30	23	0·787	48·07	+33	
Authors.	Pernicious anæmia.	M.	32	44·21	36·60	72–115	20–24	21	0·865	39·61	+7	
Authors.	Pernicious anæmia.	M.	50	41·80	37·30	80–100	18–24	44	0·830	33·53	+2	
Authors.	Pernicious anæmia.	M.	38	60·05	37·02	..	..	40	0·767	36·78	+6	

[1] Compiled by Meyer and Du Bois, *Archives of Internal Medicine*, Volume XVII, 1916.

Emaciation.

Emaciation is by no means an invariable feature of anæmia. Many patients with pernicious anæmia and chlorosis, for example, are quite plump in spite of anæmia. Again, many diseases which produce anæmia also produce emaciation. On the other hand, it has been found experimentally that animals exposed to severe and prolonged oxygen lack rapidly lose weight (Campbell[10][11]) and the same thing is observed in human beings at very high altitudes

(Ruttledge[68]). The causes of this loss of weight are not quite clear. Diminished consumption of food, due to anorexia, increased metabolism (not in the severest forms of oxygen lack) and dehydration may all play a part. In addition, oxygen lack and carbon dioxide excess have an effect upon carbohydrate metabolism, and probably indirectly upon fat metabolism. Asphyxia causes the disappearance of glycogen from the liver, the mobilization of the fat in the fat depots, and the deposition (infiltration) of this fat in the viscera, especially the liver. All these facts suggest that emaciation may be one of the results of very severe anæmia.

REFERENCES TO THE SYMPTOMS AND SIGNS OF ANÆMIA.

(1) G. W. Balfour: "Clinical Lectures on Diseases of the Heart and Aorta", 1897.

(2) D. Ball: "Changes in the Size of the Heart in Severe Anæmia, with Report of a Case", *American Heart Journal*, Volume VI, 1931, page 517.

(3) J. Barcroft, C. A. Binger, A. V. Bock, J. H. Doggart, H. S. Forbes, G. Harrop, J. C. Meakins and A. C. Redfield: "Observations upon the Effect of High Altitude on the Physiological Processes of the Human Body, Carried Out in the Peruvian Andes, Chiefly at Cerro de Pasco", *Transactions of the Royal Society of London*, Series B, Volume CCXI, 1923, page 351.

(4) J. Barcroft: "The Respiratory Function of the Blood". Part I: "Lessons from High Altitudes", 1925.

(5) H. L. Blumgart, S. L. Gargill and O. R. Gilligan: "Studies on the Velocity of Blood Flow", *Journal of Clinical Investigation*, Volume IX, 1930, page 685.

(6) K. Bohland: *"Über den respiratorischen Gaswechsel bei verschiedenen Formen der Anämie"*, *Berliner klinische Wochenschrift*, Volume XXX, 1893, page 417.

(7) W. M. Boothby and I. Sandiford: "Summary of Basal Metabolism Data on 8,614 Subjects, with Especial Reference to the Normal Standards for the Estimation of the Basal Metabolic Rate", *Journal of Biological Chemistry*, Volume LIX, 1922, page 783.

(8) W. M. Boothby and I. Sandiford: "Basal Metabolism", *Physiological Reviews*, Volume IV, 1924, page 69.

(9) R. C. Cabot: "Facts on the Heart", 1926.

(10) J. A. Campbell: "Note on Some Pathological Changes in the Tissues during Attempted Acclimatisation to Alterations of O_2 Pressure in the Air", *The British Journal of Experimental Pathology*, Volume VIII, 1927, page 347.

(11) J. A. Campbell: "Further Observations on Oxygen Acclimatisation", *Journal of Physiology*, Volume LXIII, page 325.

(12) W. S. Cann: "Calorimetry in Medicine", 1924 (Medicine Monographs).

(13) Chauveau: *Gazette Médicale de Paris*, Volume XIII, 1858, pages 247, 261, 273, 312, 340, 355, 482, 581, 592. Papers summarized in Schmidt's *"Jahrbücher der in- und ausländischen gesammte Medizin"*, Volume CII, 1859, page 228.

(14) C. F. Coombs: "A Note on the Cardiac Symptoms of Pernicious Anæmia, with Particular Reference to Cardiac Pain", *The British Medical Journal*, Volume II, 1926, page 185.

(15) D. J. Corrigan: "Inquiry into the Causes of 'bruit de soufflet' and 'frémissment cataire'", *The Lancet*, Volume II, 1828-1829; pages 1 and 33.

(16) C. G. Douglas, J. S. Haldane, Y. Henderson and E. C. Schneider: "Physiological Observations Made on Pike's Peak, Colorado, with Special Reference to Adaptation to Low Barometric Pressures", *Proceedings of the Royal Society of London*, Series B, Volume CCIII, page 185.

(17) D. Du Bois and E. F. Du Bois: "Clinical Calorimetry", *Archives of Internal Medicine*, Volume XVII, 1916, page 863.

(18) E. F. Du Bois: "Basal Metabolism in Health and Disease", 1927.

(19) F. Eberstadt: *"Über den Einfluss chronischer experimenteller Anämien auf den respiratorischen gaswechsel"*, *Archiv für experimentelle Pathologie und Pharmakologie*, Volume LXXI, 1912-1913, page 329.

(20) H. E. Eppinger, F. Kisch and H. Schwarz: *"Das Versagen des Kreislaufes"*, 1927.

(21) A. H. Elliott: "Anæmia as a Cause of Angina Pectoris in Presence of Healthy Coronary Arteries and Aorta", *American Journal of the Medical Sciences*, Volume CLXXXVII, 1934, page 185.

(22) W. M. Fletcher and F. G. Hopkins: "The Respiratory Process in Muscle and the Nature of Muscular Motion", Croonian Lecture, *Proceedings of the Royal Society of London*, Series B, Volume LXXXIX, 1917, page 444.

(23) F. R. Fraser: "Cardiac Dyspnœa", *The Lancet*, Volume CCXII, 1927, page 590.

(24) C. L. Gemmill and D. L. Reeves: "The Effect of Anoxemia in Normal Dogs Before and After Denervation of the Carotid Sinuses", *American Journal of Physiology*, Volume CV, 1933, page 487.

(25) C. L. Gemmill, E. M. K. Geiling and D. L. Reeves: "The Respiratory Effect of Prolonged Anoxemia in Normal Dogs Before and After Denervation of the Carotid Sinuses", *American Journal of Physiology*, Volume CIX, 1934, page 709.

(26) Gendrin: *"Leçons sur les maladies du cœur"*, Volume I, 1841, page 100.

(27) D. Goldstein and E. P. Boas: "Functional Diastolic Murmurs and Cardiac Enlargement in Severe Anæmias", *Archives of Internal Medicine*, Volume XXXIX, 1927, page 226.

(28) E. Grafe: *"Die steigerung des Stoffwechsels bei chronischer Leukämie"*, *Archiv für klinische Medizin*, Volume CII, 1911, page 406.

(29) E. Grafe: *"Beitrage zur Kenntnis der Kompensationseinrichtungen bei chronischen experimentellen Anämien"*, *Münchener medizinische Wochenschrift*, Volume LI, 1912, page 2840.

(30) E. Grafe: *"Zur Kenntnis des Gesammtstoffwechsels bei schweren chronischen Anämien des Menschen"*, *Deutsches Archiv für klinische Medizin*, Volume CXVIII, 1915, page 148.

(31) H. C. Gram: "On the Causes of the Variations in the Sedimentation of the Corpuscles and the Formation of the Crusta Phlogistica (Buffy Coat) on the Blood: A Preliminary Investigation", *Archives of Internal Medicine*, Volume XXVIII, 1921, page 312.

(32) G. L. Gulland and A. Goodall: "The Blood", 1914.

(33) N. B. Gwyn: "Angina Pectoris Occurring in the Course of Certain Anæmias", *Canadian Medical Association Journal*, Volume XXVI, 1932, page 26.

(34) T. R. Harrison: "Failure of the Circulation", 1935.

(35) H. E. Hering: *"Die Karotissinusreflexe auf Herz und Gefässe"*, 1927.

(36) J. B. Herrick: "Combination of Angina Pectoris and Severe Anæmia", *American Heart Journal*, Volume II, 1927, page 351.

(37) C. Heymans, J. J. Bouckaert, P. Regniers: *"Le sinus carotidien"*, 1933.

(38) Heynsius: *Nederlandischer Lancet*, Series III, Volume 1854, pages 13, 20.

(39) Heynsius: *"Über die Ursachen der Töne und Geräusche im Gefässystem"*, 1878, page 23.

(40) A. V. Hill, C. N. H. Long and H. Lupton: "Muscular Exercise, Lactic Acid and the Supply and Utilization of Oxygen", Parts I-III, *Proceedings of the Royal Society of London*, Series B, Volume XC, 1924, page 438; Parts IV-VI, *ibidem*, Volume XCVII, page 84.

(41) R. W. G. Hingston: "Physiological Difficulties in the Ascent of Mount Everest", *Geographical Journal*, Volume LXV, 1925, page 4.

(42) M. Kahn and J. Barsky: "Studies of the Chemistry of Pernicious Anæmia", *Archives of Internal Medicine*, Volume XXIII, 1919, page 334.

(43) F. Kisch: *"Experimentelle und klinische Untersuchungen über das Verhalten des Blutfibrinogens bei pathologischer Zuständen"*, *Klinische Wochenschrift*, Volume II, 1923, page 1452.

(44) Fr. Kraus and Fr. Chvostek: *"Über den Einfluss von Krankheiten besonders von anämischen Zuständen, auf den respiratorischen Gaswechsel"*, *Zeitschrift für klinische Medizin*, Volume XXII, 1893, page 449.

(45) E. M. Landis: "Micro-Injection Studies of Capillary Permeability. III: The Effect of Lack of Oxygen on the Permeability of the Capillary Wall to Fluid and to the Plasma-Proteins", *American Journal of Physiology*, Volume LXXXIII, 1927-1928, page 528.

(46) W. G. Lennox, F. A. Gibbs and E. L. Gibbs: "Relationship of Unconsciousness to Cerebral Blood Flow", *Archives of Neurology and Psychiatry*, Volume XXXIV, 1935, page 1001.

(47) T. Lewis, G. W. Pickering and Rothschild: "Intermittent Claudication", *Heart*, Volume XV, 1929-1931, page 359.

(48) C. Lundsgaard and D. D. van Slyke: "Cyanosis" (Medicine Monographs), *Medicine*, Volume II, 1923, page 1.

(49) J. S. McLester, M. T. Davidson and B. Frazier: "Blood Fibrin Changes in Various Diseases, with Especial Reference to Diseases of the Liver", *Archives of Internal Medicine*, Volume XXXV, 1925, page 177.

(50) A. Magnus-Levy: *"Der Einfluss von Krankheiten auf den Energiehaushalt im Ruhezustand"*, *Zeitschrift für klinische Medizin*, Volume LX, 1906, page 177.

(51) J. C. Meakins and H. W. Davies: "The Respiratory Function in Health and Disease", 1925.

(52) E. Meulengracht, P. Iversen and F. Nakazawa: "Pernicious Anæmia: Edema and Reduction in Excretion of Water", *Archives of Internal Medicine*, Volume XLII, 1928, page 425.

(53) O. Meyerhoff: *"Die Energieumwandlungen im Muskel. V: Milchsäurebildung und mechanische Arbeit"*, *Archiv für die gesammte Physiologie*, Volume CXCI, 1921, page 128.

(54) A. L. Meyer and E. F. Du Bois: "The Basal Metabolism in Pernicious Anemia", *Archives of Internal Medicine*, Volume XVII, 1916, page 965.

(55) A. Blackhall-Morison: "The Musculature of the Aorta and the Cardiac Valves, Anatomically, Physiologically and Clinically Considered", *Edinburgh Medical Journal*, Volume XXX, 1923, "Transactions of the Medico-Chirurgical Society", page 149.

(56) R. Nuzum and J. C. Herrick: "Angina Pectoris", *The Journal of the American Medical Association*, Volume LXX, 1918, page 67.

(57) D. Noël Paton: "On the Action of Valves of the Mammalian Heart", *Transactions of the Royal Society of Edinburgh*, Volume CLXXXIV, 1892, page 179.

(58) D. Noël Paton: "On the Action of the Valves of the Mammalian Heart", *Reports of the Royal College of Physicians Laboratory, Edinburgh*, Volume IV, 1892, page 36.

(59) F. W. Peabody: "Clinical Studies of the Respiration. III: A Mechanical Factor in the Production of Dyspnœa in Patients with Cardiac Disease", *Archives of Internal Medicine*, Volume XX, 1917, page 435.

(60) F. W. Peabody and J. A. Wentworth: "Clinical Studies of the Respiration. IV: The Vital Capacity of the Lungs and its Relation to Dyspnœa", *Archives of Internal Medicine*, Volume XX, 1917, page 443.
(61) F. W. Peabody and C. C. Sturgis: "Clinical Studies of the Respiration. VII: Effect of General Weakness and Fatigue on the Vital Capacity of the Lungs", *Archives of Internal Medicine*, Volume XXVIII, 1921, page 501.
(62) J. P. Peters, A. J. Eisenman and H. A. Bulger: "The Plasma Proteins in Relation to Blood Hydration. I: In Normal Individuals and in Miscellaneous Conditions", *Journal of Clinical Investigation*, Volume I, 1925, page 435.
(63) M. v. Pettenkofer and C. Voit: *"Über den Stoffverbrauch bei einem leukämischen Manne"*, *Zeitschrift für Biologie*, Volume V, 1869, page 319.
(64) G. W. Pickering and E. J. Wayne: "Observations on Angina Pectoris and Intermittent Claudication in Anæmia", *Clinical Science*, Volume I, 1934, page 305.
(65) W. D. Reid: "The Heart in Pernicious Anemia", *The Journal of the American Medical Association*, Volume LXXV, 1923, page 534.
(66) Fr. Rolly: *"Über den respiratorischen Gaswechsel bei chronisch anämischen Zuständen"*, *Deutsches Archiv für klinische Medizin*, Volume CXIV, 1914, page 605.
(67) A. H. Rowe: "Refractometric Studies on Serum Proteins in Cardiac Decompensation, Diabetes, Anæmias and other Chronic Diseases", *Archives of Internal Medicine*, Volume XIX, 1917, page 354.
(68) H. Ruttledge: "Everest 1933", 1934.
(69) F. Savart: *"Mémoire sur la constitution des veines liquides lancées par les orifices circulaires en mince paroi"*, *Annales de chimie et de physique*, Series 2, Volume LIII, 1833, page 337.
(70) C. F. Schmidt: "Carotid Sinus Reflexes to the Respiratory Centre. II: Attempt at Evaluation", *American Journal of Physiology*, Volume CII, 1932, page 119.
(71) E. C. Schneider and D. Truesdell: "Circulatory Responses of Man to Sudden and Extreme Anoxemia", *American Journal of Physiology*, Volume LXV, 1923, page 379.
(72) S. Seliadurai and S. Wright: "Mode of Action of Respiratory Stimulants. I: Mode of Action of Oxygen Lack", *Quarterly Journal of Experimental Physiology*, Volume XXII, 1933, page 233.
(73) H. J. Stewart, N. F. Crane and J. E. Deitrick: "Studies of the Circulation in Pernicious Anemia", *Journal of Clinical Investigation*, Volume XVI, 1937, page 431.
(74) C. S. Sturgis, F. W. Peabody, F. C. Hall and F. Fremont Smith: "Clinical Studies of the Respiration. VIII: The Relation of Dyspnœa to the Maximum Minute Volume of Pulmonary Ventilation", *Archives of Internal Medicine*, Volume XXIX, 1922, page 236.
(75) O. Thiele and O. Nehring: *"Untersuchungen des respiratorischen Gaswechsels unter dem Einfluss von Thyroideapräparaten und bei anämischen Zuständen des Menschen"*, *Zeitschrift für klinische Medizin*, Volume XXX, 1896, page 41.
(76) E. H. Tompkins, H. H. Brittingham and K. Drinker: "The Basal Metabolism in Anemia, with Especial Reference to the Effect of Blood Transfusion on the Metabolism of Pernicious Anemia", *Archives of Internal Medicine*, Volume XXIII, 1919, page 441.
(77) S. Wright: "Further Observations on the Mode of Action of Oxygen Lack on Respiration", *Quarterly Journal of Experimental Physiology*, Volume XXVI, 1936, page 63.
(78) N. Zuntz, A. Löwy, F. Müller and W. Caspari: *"Höhenklima und Bergwanderungen in ihrer Wirkung auf den Menschen"*, 1906.

ACCIDENT INJURIES AND COMPENSATION.[1]

By F. V. SMITH, K.C.

Introduction.

THIS is a situation of unusual advantage for me, for I usually speak in places of violent disputation. But tonight, having eaten and drunk with friends, I can converse with them in the same happy circumstances. I join with Sir Henry Newland in offering to the governing authorities of this society my congratulations upon instituting this pleasant mode of exchanging views upon topics which are of common interest to us. It is a good thing to step aside from time to time out of the traffic of our daily occasions to contemplate in friendly intercourse the means by which each of our professions may, by being more closely knit, be of still greater service to the community. They are both indispensable agents in the administration of justice. One at least of the professions represented has in its custody and control the sacred trust of public health, and the other has no less a sacred trust in the administration of justice between fellow citizens of the King. Between them they make a great contribution to the orderly and happy lives of the subjects of the King, and their closer cooperation can scarcely fail to produce a great public benefit.

The subject chosen is one of abounding interest to both professions, treated from the medical side. From the juristic side its interest, I fear, is unilateral, confined exclusively to the legal profession. For the difference between these two great professions lies in part in this, that whenever they meet, in their common search after truth, any instruction or assistance in medical science cannot possibly come amiss to the lawyer and must be of service to him. But the converse is not true. The doctor has only a strictly limited interest in juridical philosophy, nor, except so far as his professional behaviour and duty are dictated by law and enforced by its sanctions, need he know anything about it. His interest does not rise above that of any other citizen, except in special departments where legal philosophy is disputed by medical theory. And even there the dispute is more ostensible than real, and, for the most part, arises from failure to understand that the lawyer and the doctor are accustomed by training to regard a common subject matter on the one hand objectively and on the other hand subjectively. This view can be best illustrated by reference to the conflict of opinions existing between them in matters affecting criminal responsibility among persons with disordered minds. The doctor, in the presence of a mind which is liable to be overtaken by an uncontrollable impulse, denies that it is a voluntary agent of a man's will. His test is the power of resistance resident in the mind of the patient, and the mercy of his great art selects him as a subject for treatment and not for punishment. The lawyer, on the other hand, regarding him as a unit of the corporate body we know as society, insists that civic safety demands his punishment if his mind is so far normal that it can distinguish right from wrong. Here are irreconcilable aspects of the same subject. Both views are right according to the approach you make to the problem. In one sense it is unjust to condemn a man who is guilty of an act known to him to be wrong but the perpetration of which he was powerless to resist. In another sense, it is just for the defence of the community that a mind which knows the quality of his act should pay the price of his crime. *Salus populi suprema lex.*

The subject at hand is, however, one on which there is no duality of opinion between the professions. Here the respective departments of law and medicine are insulated. The lawyer accepts the preponderating testimony of the medical profession. The medical man needs to know very little of the law of the subject to which he contributes his evidence. Hence it is that from the purely legal

[1] Read before a meeting of the Medico-Legal Society of South Australia.

side it is difficult to say much that will be of interest or profit to the doctor, for the fullest examination of underlying principles will be as devoid of interest as of service to him. It will thus be seen that though the subject is one strictly within the territory of medical jurisprudence, its treatment from the juristic side has little interest for those outside the legal profession.

Some obversations on compensation may, however, interest both professions.

I hope that everything I say will be understood to be said in the friendliest sense. I am not here to make admonitions, but I have set down some reflexions of mine based on a not inconsiderable experience. They are not intended in the least to be a criticism of the medical profession, though I hope they may in some part help to smooth out the corrugations of our common path.

The Significance of Compensation.

In the first place it must be remembered that the word "compensation" is a word of wide significance at law. It includes damages at common law for wrongful injury, the assessed value of expropriated property, and several kinds of statutory satisfaction for injury suffered. In some cases, as in ordinary torts or wrongs, the damages may be quite at large; in other cases, as in workmen's compensation, they may be strictly regulated on an insurance basis without any reference to compensation in the ordinary sense of that term for the injury suffered. This is so because employers' liability is quite a modern development of law. The principle of damages began in primitive law as a substitute for personal retaliation; for example, weregild was the price of a man's life—no doubt descended from *lex talionis*. Before the *Employers' Liability Act*, 1884, the field in which the workman could recover from his master was extremely narrow. In mediæval times, owing to the influence of Roman law, the relations between master and servant were for the most part governed by the law of status. But about the onset of the Industrial Revolution, and with the growth of machinery, actions by servants against their employers for personal injury began to multiply. But the actions were always conceived in tort, never in contract, and the result was that in order to succeed the servant always had to show a personal actual fault, that is, breach of duty to take care, on the part of the employer. Even when he did so he was liable to be defeated by showing that he was guilty of contributory negligence, or that he had assumed the risk incident to the employment, or that the injury had been caused by a fellow servant, the infernal doctrine of common employment. To put an end to, or at least to qualify, these defences the *Employers' Liability Act*, 1884, was introduced, but again the statute treated the liability as arising *ex delicto* and not being incident to the contract of employment. Further, it limited the amount of damages which could be recovered to the estimated earnings of three years preceding the injury. This was an arbitrary abrogation of the court's power to award a sum commensurate with the injury, including medical expenses and personal suffering.

There followed the *Workmen's Compensation Act*, 1900, which has developed into the *Workmen's Compensation Act*, 1932. In this type of legislation the right to recover is entirely wrested from the domain of tort and is annexed as a statutory incident to the contract of service itself. The recoverability of compensation depends upon the employment, not upon some delict in the master or his other employees. It is now a contractual, not a delictual, responsibility, fixed by statute upon every employer. In each of these three different types of employers' liability a different calculus is applied for the ascertainment of the compensation to be paid.

At common law, and under the *Employers' Liability Act* within its pecuniary orbit, in the relatively few instances in which a servant could recover at all, the compensation was assessed upon principles precisely the same as in accidents between strangers. The medical witness in such a case requires to address himself to a far wider field of injury than in workmen's compensation cases. The court, within certain limits, strives to bring about as nearly as it can a *restitutio in integrum*. This necessarily requires in accident cases a very meticulous and careful survey of the victim's injuries, including those of a psychical kind, such as shock, pain and suffering, and as exact a forecast of future or permanent disabilities as skill aided by experience and judgement can make.

Expert Evidence.

It is an admitted infirmity of the human mind that it is liable to take sides. One of the greatest difficulties that beset the greatest of judges is to reserve finality till all is heard, and it was one of the French essayists who spoke of the family for its units, the partisanship of the "hierarchy of patriotism"—the partisanship of districts, of cities, of nations. It is not surprising, therefore, that it should be revealed in the opinion evidence of experts. This partisanship is no reflexion upon the probity of the expert; it is a species of unconscious advocacy, and is noticed in lay witnesses as well. Nowhere is this weakness more perceptible than in medical accounts of injuries, whether with a view to heightening or lowering damages. The evidence of medical experts is extremely helpful to the administration of justice; but it is the reverse of edifying to find the estimates of equally conscientious and competent men varying to the point of contradiction.

A very great and illustrious lawyer, Lord Bramwell, was often quoted as saying that witnesses fall into three divisions, namely, liars, damned liars and expert witnesses. And this apothegm is much in vogue when we have not got experts to assist us. It is not generally known, but it is a fact, that Bramwell, years afterwards Lord, was asked to repeat his brilliant epigram. He himself belonged to a very distinguished family. His elder brother was a very distinguished surgeon in the Indian Civil

Service. His younger brother, Frederick, was an eminent civil engineer. His services were in great demand in cases of compulsory resumption and compensation as an expert witness. When Lord Bramwell was asked to repeat his acid generalization he did so by restating the three divisions, liar, damned liar and expert witnesses, and then he added, thoughtfully, "then there is my brother Frederick".

I am convinced from my experience in courts that medical witnesses, for some reason I shall never know, all resent cross-examination. I cannot say how much that has amazed me. Cross-examination is an indispensable instrument for the investigation of truth. It is a gross mistake to suppose that its administration implies a reflexion either on the skill or the probity of the witness. Pressure sometimes there is. But pressure is not the same thing as hostility, and the witness ought to remember that the cross-examiner is simply advancing to the witness the contrary views and opinions of the experts who are instructing him and in whom he is bound to believe. Nor should he react to cross-examination in a partisan spirit. It is his solemn duty to assist the court irrespective of the side which calls him, and the attitude of the offended angel which he sometimes wears is much more properly worn by his patient, is it not? He should not be jealous of his learning nor impatient if his art is for the time being invaded by the uninitiate. No barrister ever pretends that his examination is based on any wide learning of what is the peculiar art of a doctor. I protest, I hope on behalf of both professions, upon statements I have seen in medical journals in which cross-examination is held up to be an ignorant attempt to hinder the medical witness in his effort to demonstrate the truth. If medical gentlemen would remember that they are, as we are, mere servants of the law, although in different ways, each of us striving to attain the noble end of truth, there will be no need for acrimony or heat.

Justice is best achieved by the attrition of two minds, as nearly balanced as may be, grinding out the truth between them, and experience has shown that that is the best way of elucidating the problems of the most complex case. A doctor who comes into the witness box should, remembering the sanctity of his profession, be clothed in the garments of neutrality. So far from resenting cross-examination, he should welcome it. I am not overlooking that a doctor has sometimes to put up with a great deal. I have heard questions asked which might well try the temper of the most patient man: questions born of ignorance; questions put with a good deal of involution and obscurity; but I do not doubt that as a rule the judge is able to intervene to assist the medical man.

I have said that the courts aim at a *restitutio in integrum*; but this does not mean that they attempt to assess exactly in terms of money the loss of a limb or the agonies of post-operative recovery.

"How is anybody to measure pain and suffering in moneys counted?" asks Lord Halsbury. In cases of contract you can often demonstrate mathematically the precise loss suffered. In torts this is not possible, and the only measure capable of enunciation is, after consideration of all the circumstances, including pain and suffering and diminished enjoyment of life, to award a fair compensation for the delict of the defendant. The compensation aims to be a solatium, not a market price.

In workmen's compensation, the assessment of compensation excludes heads of damage, such as shock or pain and suffering. Here the effort is to ascertain, with the assistance of medical witnesses, the extent of partial incapacity or the existence of total incapacity. The amount of compensation is fixed in the case of death at a maximum of £600 with a minimum of a sum equal to his earnings for the preceding three years, or £400, whichever is greater.

In the case of total or partial incapacity for work the amount of compensation is also fixed by reference to his weekly earnings over the twelve months preceding the injury.

In these cases the investigation into compensation is simplified by the fixation of amounts in certain specified injuries enumerated in the statute. This exonerates the medical witness from any careful testimonial survey of the injuries of the patient beyond proof of the particular injury to which the statute applies. But this does not mean that, within the limits of his task, his job is lightened.

Medical Referee.

One of the most important and difficult of professional duties assigned to the medical profession under the *Workmen's Compensation Acts* is the certification of the condition of an injured workman and his fitness for employment. The court has power to appoint a medical referee for this purpose after the man has been examined by a medical practitioner for the workman and by another for the employer. The referee may also be empowered to certify as to whether or to what extent incapacity exists. The certificate, when given, is conclusive against the workman and the employer, and the courts presume in its favour that the medical referee has applied his mind to all the issues raised in the submission and that he has made all such relevant inquiries and investigations as are necessary to support the certificate. I need not emphasize the high degree of responsibility which is thus vested in the medical practitioner, nor the need for the most scrupulous care in discharging it. An error of judgement here may irreparably injure an afflicted workman or irrevocably settle upon the employer an unjust and permanent liability.

Suicide and Mental Derangement.

It gives some idea of the width of the net spread by the *Workmen's Compensation Act* when I say that an employer can be held liable to pay compensation when his workman suffers an accident in the course of his employment which produces some mental derangement leading to suicide. Here both the lawyer and the doctor are caught in a

vortex of elusive subtleties. For first, contrary to some medical opinion, the law does not deduce insanity from suicide. It takes into account the fact that the *tædium vitæ* of the Stoics and Epicureans can coexist with a robust intellectual constitution; and from the common terms of their policies insurance companies seem to recognize the some thing. An example of a premeditated suicide is furnished by the case of Bevan *v.* Lancaster Steam Collieries, where a workman who suffered great pain from incurable injuries determined upon suicide and left a letter in which he explained in a rational and coherent way his reasons for his act. Here the claim failed because his act was the reasoned act of a competent person and the chain of causation between the injury and the death was interrupted by a *novus actus interveniens*.

There must be evidence of actual mental derangement resulting directly from the injury and leading directly to the act of suicide. It is here that the factual difficulties are most encountered. A mere depression of spirits, such as is frequently experienced, will not suffice. Such depression may lead intelligent thought to the conclusion that escape from present evils is worth the price of self-destruction. Yet it must be admitted that depression of spirits and melancholia are only expressions of the degree of mental suffering. And if depression of spirits is a direct result of the accident and a direct cause of the suicide, it is difficult to see why compensation should not be given to the dependants. A depression of spirits so acute as to lead to suicide is to some extent at least one form of mental derangement, and it seems logical to say that, when suicide is a direct product of the derangement and the derangement is immediately traced to the injury, there is an uninterrupted chain of causation. Nevertheless, there exists in law a broad distinction between mental derangement arising directly out of the accident and mental derangement caused indirectly from brooding over the injuries received.

The mental derangement need not be so acute as to render the patient a certifiable lunatic. Here there is no question of insanity in the legal sense; the law, on this branch, is satisfied with the medical view. This involves no antinomy in jurisprudence. The criterion of insanity in crime is peculiar to that branch of jurisprudence so that the accused may, if he satisfies it, receive the absolution of the law. No such considerations arise in this case. Here the inquiry is not into the moral responsibility of the deceased workman, but into the question whether his suicide can be referred back directly to the injury which he suffered.

The Need for Laws of Compensation.

The extraordinary, even fantastic, example of an employer being held liable for his workman's suicide draws attention to the need of some juridical basis to justify the imposition of burdens created by the act of another. This head of jurisprudence has been singularly neglected in England, where the innovations in the common law first found appearance. But it has been tenaciously explored on the continent of Europe and in the United States of America. The most widely accepted theory at the present time is the theory of *risque professionel*, or "trade risk". It is based upon the premise that industrial accidents arise for the most part from the nature of modern industry itself. Risks attend every enterprise, whether to plant, machinery, finance, or the human element which operates it. It is a commonplace of economics that the burden of all risks to inanimate assets employed in an enterprise should be borne by the industry. Why not, therefore, the risks to the more valuable and no less indispensable animate and human plant of the industry? But industry consists both of capital and labour, and accordingly both should share the loss, since both reap the rewards of the enterprise—capital receiving profits, labour recovering wages. This theory at once explains and defends the awards of compensation which are admittedly incommensurate with the pain and injury suffered and much below the amount which would be deemed adequate were the injury attributable to the wrongful act of a party. This theory, which makes loss by injury fall upon the constituents of industry, capital and labour, is not so novel as might be expected. The principle of making a common peril of a common venture is no stranger to the common law, as the doctrine of marine average shows. An excellent example of the theory is cited in a luminous article contributed to the *Southern California Law Review* by Mr. James Pike, a member of the California Bar and Sterling Fellow of the Yale University School of Law.

> An engineer is in charge of an engine, one of the cylinder heads of the engine blows out; the engine is repaired; and the cost of such repair is charged to the business as an incident to such business; but at the time the cylinder head blows out the engineer's leg is badly burned and scalded by the escaping steam, and the loss of time occasioned by the man's injured leg is ignored unless he enforces his claim in the courts. If he sees fit to demand reparation by process of law he is fought like an enemy. In justice his loss should be paid for, the same as the loss on the engine.

And it was presented in more stately diction by the late Mr. Justice Holmes:

> It is reasonable that the public should pay the whole cost of producing what it wants, and a part of that cost is the pain and mutilation incident to the production. By throwing that loss upon the employer in the first instance we throw it upon the public in the long run, and that is just.

This theory gives to the legal profession a deeper insight into the principles which underlie this important branch of law. It is a far cry from this remedial statute to the barbarous Statute of Labourers of the first Edward's day. In its enlightened operation by lawyers and in the cooperation of the medical profession, the two great professions are in possession of a field in which a great public service can be done and in which not the least of our rewards is the consciousness of having advanced the frontiers of humanity.

NOTES ON THE CASE FOR ELECTROTHERAPY.

By E. P. DARK, M.C., M.B., Ch.M.,
Katoomba.

IT should not now be necessary to state a case for electrotherapy, but Australia, and more especially Sydney, still appears to be many years behind Europe and America in using the varied aids this branch of the healing art has to offer.

Unfortunately a large proportion of the senior members of our profession in Sydney have neither knowledge of nor interest in any form of electrotherapy, except perhaps diathermy, which they occasionally use inefficiently as a forlorn hope. I can see no excuse for this, whether it be due to mental inertia or to an unjustified extreme of scepticism. The facts have been published again and again, in books and in journals.

To give one instance, H. E. Stewart, in 1923, published his "Diathermy with Special Reference to Pneumonia"; his case records were very convincing. A brief summary of his results was given in the first edition of my "Diathermy in General Practice" in 1930 and in the second edition in 1934; the results have been published again in the third edition of E. P. Cumberbatch's "Diathermy". Inductothermy now offers an infinitely simpler technique and a much more penetrating form of heat; but has any senior physician in any great metropolitan hospital thought it worth while to make any further investigation?

A year or so ago an article on the treatment of facial paralysis appeared in THE MEDICAL JOURNAL OF AUSTRALIA; no mention was made of the use of infra-red rays, yet in the second edition (1933) of Annandale Troup's "Therapeutic Use of Infra-Red Rays" it was claimed that these rays were practically specific for Bell's palsy. Since I began to use infra-red rays, I have had three patients with complete facial paralysis, and all recovered fully in from seven to ten days with no treatment but infra-red rays. In a recent article in the same journal the treatment of chilblains is discussed, but no mention is made of ultra-violet radiation; yet this is valuable in obstinate cases, and is mentioned by Robert Aitken in "Ultra-Violet Radiations and their Uses" (1931), and my own experience with this form of treatment has been very satisfactory.

Last year, at a meeting in Sydney at which many senior members of the profession were present, it was noted as a new and surprising observation that diathermy was very effective in the treatment of gonococcal arthritis. Cumberbatch, in the first edition of his "Diathermy", published in 1921, pointed this out. Such a state of ignorance would be funny if it were not a disgrace.

Some time ago both Dr. R. W. Richards, of Blackheath, and Dr. A. Alcorn, of Katoomba, referred to me patients with fractures of the shaft of the tibia which had failed to unite; in each case firm bony union followed five weeks' treatment with ultra-violet rays and diathermy. The results were confirmed by X ray examination, and one of Sydney's leading orthopædic surgeons saw the series of X ray photographs; he had never tried the method.

Recently a patient who had an obscure pain in the shoulder was sent for an opinion to a very senior consulting physician in Sydney. The part of the report which dealt with treatment said that some form of heat might help. Hot foments, "Poulticine", hot air treatment, diathermy, infra-red radiation, inductotherm and short wave therapy all supply forms of heat, and all of them give different depths of heat penetration. Several of them heat different tissues to varying degrees. The only reasonable conclusion is that this very senior consultant simply did not know that fact. The advice was about as useful as if, in the case of a patient suffering from an obscure endocrine deficiency, the suggestion were that some form of glandular preparation might help.

Last year a young man came to me from Sydney. Several months previously he had sustained a fracture near the elbow joint. After union had occurred there had been considerable limitation of movement, and the patient had been sent by the chief referee of his insurance company for hot air treatment and massage—hot air treatment in 1936, be it noted. He made fair progress until he could extend his forearm to an angle of 120°, and there progress ceased during five weeks of treatment. When I saw the patient this was the condition of his arm; the forearm had wasted by 1·9 centimetres. I gave the wasted muscles daily stimulation with the Bristow coil, and then heated the elbow joint for half an hour with the inductotherm and gently manipulated it. In four weeks the patient had full extension of the forearm and no wasting.

Early in 1935 I treated a man, aged sixty years, for a very severe strain of the internal lateral ligament of his knee joint. There was much fluid in the joint, which was five centimetres larger in circumference than the other. The patient remained in bed for about a month and was treated with diathermy. When he could walk he came to my rooms for inductotherm treatment. Seven weeks after the accident, as he was still not cured, the insurance company sent him to Sydney to see an orthopædic surgeon.

The report stated that the circumference of the right joint was 2·2 centimetres greater than that of the left; movement of the joint was from 175° to 80°; there were 1·2 centimetres of atrophy of the quadriceps. The consultant considered that this limitation of full extension was due to a persistent displacement of a torn internal semilunar cartilage, and that after the length of time that had elapsed spontaneous or manipulative reduction was not likely to be effective. He recommended operation on the joint and the removal of the internal semilunar cartilage. The patient's convalescence, he thought, would probably occupy a period of three months.

The patient was not anxious for operation, and the insurance company very reasonably allowed me to carry on for three weeks, which I did, at the patient's earnest request, but without much optimism, as I agreed with the orthopædic surgeon's summing up. However, he returned to work, with quite an efficient joint, in five weeks instead of thirteen.

A man was transferred to our hospital from Sydney with a history of having severely strained the lumbar muscles. The insurance company, on the advice of its medical officer,

had been sending him to a masseur for diathermy for several weeks, with no improvement. The patient was an intelligent man, and he described in detail the treatment given: the electrodes used were about seven by ten centimetres in size; he went three times a week and was given twenty minutes' treatment each time; his back never felt more than warm. In other words, a tri-weekly farce was played at the expense of the insurance company.

This is the kind of inefficient treatment that brings electrotherapy into ill repute; but it is solely the fault of the doctor who prescribes it without knowing how it should be applied or the range of its usefulness.

In this very small district there are available two diathermy appliances, two ultra-violet ray machines, one inductotherm, one ultra-short-wave machine, one infra-red lamp, one ionization outfit, and one Bristow coil. A few cases will show what can be done when a group of doctors realize the proper uses of electrotherapy.

A year and a half ago Dr. A. Allan referred to me a patient with *tic douloureux*; all drug treatment had failed and the slightest stimulus brought on a spasm. He had twenty-six daily treatments with the inductotherm, and from the sixth treatment there was steady improvement. After the twenty-sixth treatment he was quite relieved. I saw the patient two months ago, and he was still free from pain.

Dr. R. W. Richards, of Blackheath, referred to me a middle-aged man who was suffering from fairly advanced osteoarthritis of both knee joints, with considerable pain. He was given twenty-two treatments with the inductotherm, with progressive relief of symptoms, and was finally quite comfortable.

Dr. A. H. Macintosh, of Leura, sent to me for treatment a middle-aged man suffering from chronic sciatica; he had made no progress with ordinary methods of treatment. He was given daily treatments with the inductotherm for three weeks and was completely relieved.

Dr. Tasman Wilson, of Wentworth Falls, had an obscure but obstinate case of pain in the terminal phalanx of the great toe, which he thought might be due to a chronic periostitis. Three weeks' treatment with infra-red radiation quite freed the patient from pain, and there has been no recurrence in about two years.

Dr. C. E. North, of Lawson, has a middle-aged male patient suffering from spondylitis of the upper dorsal and cervical vertebræ; he has had two courses of treatment with the inductotherm, each of a fortnight's duration, and he has had about six months' relief from pain.

Another patient suffering from spondylitis has had three similar courses of treatment from me, and he enjoys from six to nine months' relief after each course.

Dr. G. T. Ferris, of Leura, referred to me a middle-aged lady with obstinate neuralgia of the lower jaw. He could find no cause for the pain, which was of two years' duration, but had recently become more severe and could no longer be relieved by sedatives. About three weeks' treatment with the inductotherm gave complete relief at the time, which was several months ago. There has been slight occasional return of the pain since, but it is easily controlled with aspirin.

Dr. A. Alcorn, of Katoomba, was treating a schoolboy who had sustained a fracture in the region of the elbow joint; several weeks after firm union had occurred extension was still limited to about 120° and improvement had ceased. The child was given sixteen treatments with the inductotherm, followed by gentle manipulation, and the end result was the recovery of full extension.

Dr. Allan also sent for treatment a man, aged seventy-six years, suffering from arthritis of a knee joint, which prevented him from playing golf and was threatening his enjoyment of bowls and billiards. He was given about four weeks' treatment with the inductotherm and resumed his golf with great pleasure.

In the Anzac Memorial Hospital of fifty beds, the use of electrotherapy is steadily increasing, as the medical officers observe for themselves its great range of usefulness. As many as eighteen treatments a day have been given with the infra-red lamp, providing patients with very many hours of comfort that would have been hours of pain. It has been used frequently for the relief of post-operative shock and pain with good effect.

General ultra-violet radiation, in association with local diathermy or infra-red radiation, is now used constantly in cases of fracture when union is slow in spite of satisfactory anatomical position, and the results completely justify its use.

The ionization outfit has only recently been bought, but will be used especially for areas of obstinate infection and for cases of neuritis or arthritis that are resistant to heat. I shall quote a few cases of special interest.

Dr. Alcorn had to operate on a patient, suffering from advanced pulmonary tuberculosis, for the removal of an appendix that was already ruptured. It was a forlorn hope operation, in which death on the table was more likely than not. After the operation the patient appeared to be dying; she was given infra-red radiation over the abdomen continuously throughout the night, and frequent prolonged exposures to infra-red rays during the following days. In such cases, when the patient should be disturbed as little as possible, the infra-red radiation may be given through the bed-clothes and whatever dressings are necessary. Dr. Alcorn is convinced that the treatment by infra-red radiation altered the verdict from death to life.

In a case of acute pancreatitis also, infra-red radiation was of great post-operative help; it considerably relieved suffering and possibly saved the patient's life. In another instance, in a different hospital, I observed the effect of infra-red radiation for twenty-four hours without interruption, following a severe and extensive operation. This was before I had learned that infra-red radiation could be continued for long periods without any ill effects. I gave one hour's infra-red radiation every three hours, and the observed effects were as follows. The patient's colour before infra-red radiation was ashen, but within a few minutes of the commencement of the treatment the colour improved and in five minutes was a healthy pink, although the face was completely shaded from the rays; the pulse fell from 96 to 84 and improved in quality, pain was greatly relieved, and during the night the patient fell asleep on each occasion within ten minutes of the turning on of the light.

REPRODUCTION OF SLIDES SHOWN BY Dr. R. WEBSTER AT A SPECIAL MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION ON JULY 29, 1937.

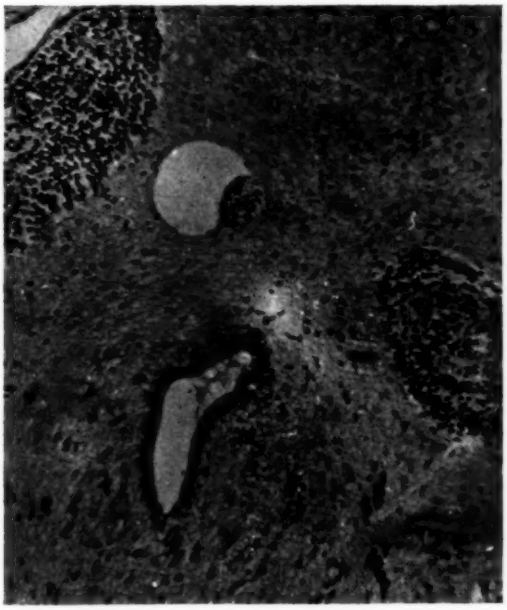


Figure I.
Low power photomicrograph showing perivascular cell collarettes.

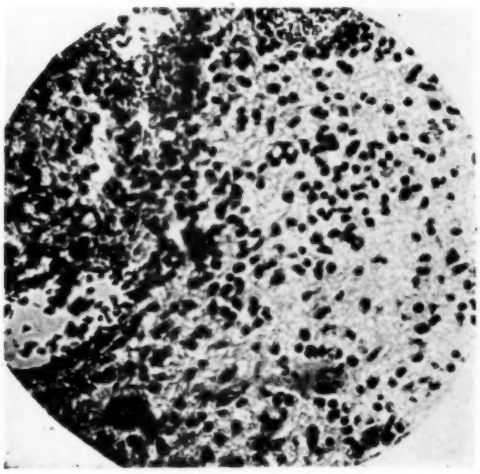


Figure II.
High power photomicrograph showing distended and engorged blood vessels and diffuse inflammatory reaction.

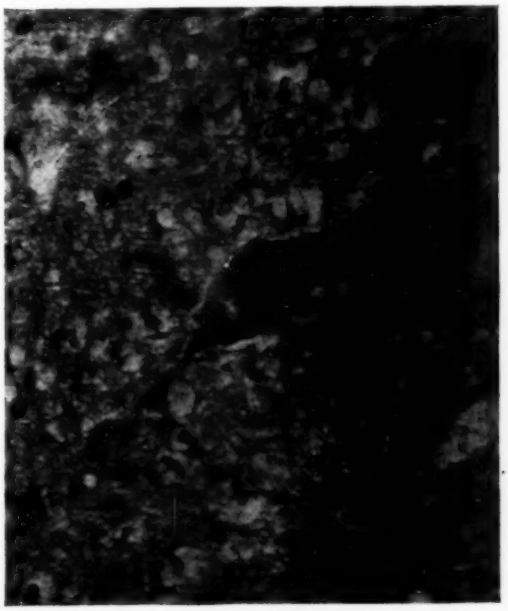


Figure III.
High power photomicrograph showing damaged and swollen ganglion cell, poor staining, loss of nucleus, swollen axis cylinder.

Figure IV.
High power photomicrograph showing the process of neuronophagia.

REPRODUCTION OF SLIDES SHOWN BY DR. R. WEBSTER AT A SPECIAL MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION ON JULY 29, 1937.

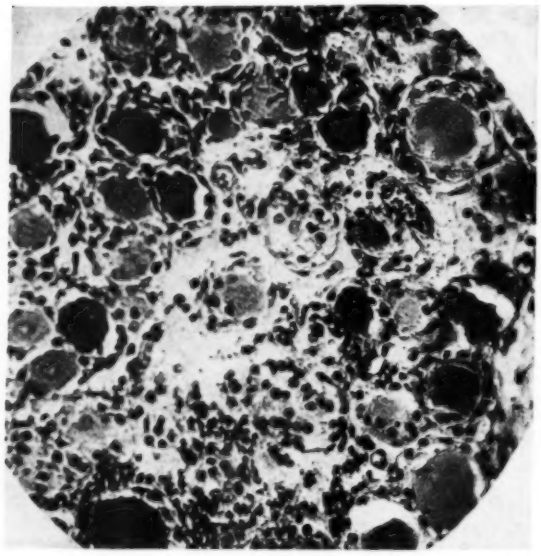


FIGURE V.

High power photomicrograph of posterior root ganglion, showing degenerative and necrotic changes in the ganglion cells, inflammatory reaction, neuronophagia.

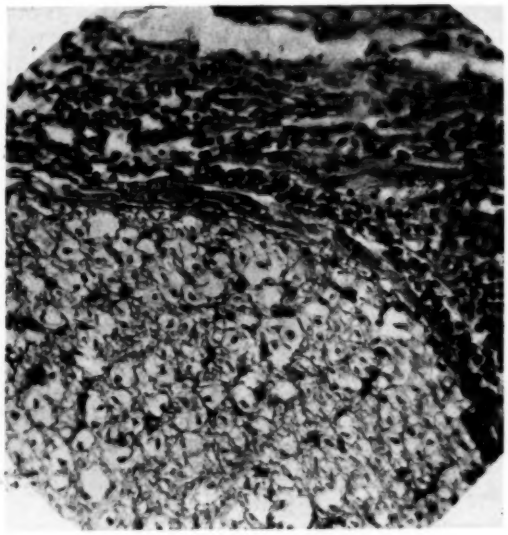


FIGURE VI.

High power photomicrograph showing meningeal reaction; aggregation of inflammatory cells in the pia-arachnoid.

ILLUSTRATION TO THE ARTICLE BY DR. GILBERT PHILLIPS.

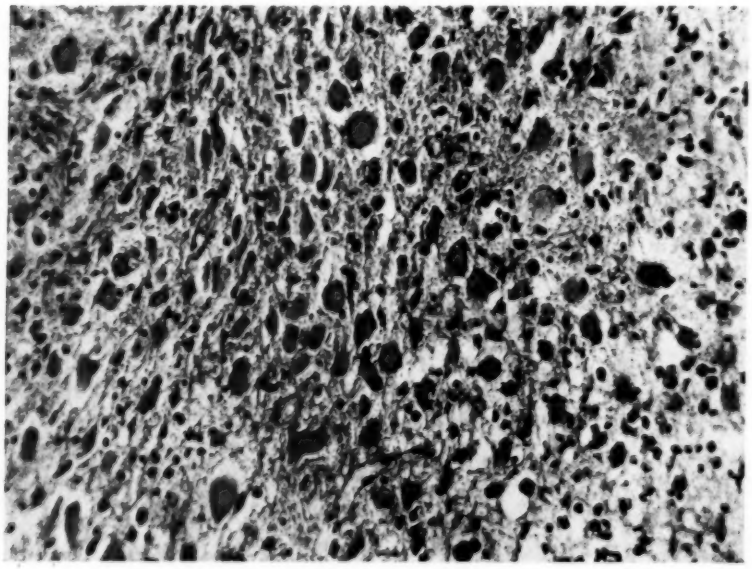


FIGURE II.

Protoplasmic astrocytoma (gemistocitic cell type). ×185.

Another of Dr. Alcorn's patients had sustained a compound comminuted fracture of both bones of the leg, tearing of the posterior tibial artery, complete dislocation of the astragalus, and much damage to the soft tissues. In two days the leg was in a very bad condition and amputation appeared to be inevitable. However, Dr. Alcorn decided to give the limb a chance by using a Schmerz hook and infra-red radiation. Progress was slow but continuous, and the patient now walks without a limp. Dr. Alcorn's considered opinion is that without infra-red radiation the leg would not have been saved.

On March 5, 1937, a woman patient of Dr. North's was admitted to hospital suffering from pneumonia; she was unconscious and had collapsed. Next morning she was given forty minutes' exposure to infra-red rays. It was noticed that her condition, which was desperate, improved while she had the lamp and grew worse when it was taken away. Dr. North suggested to me that the lamp should be left burning constantly while the patient needed it, and she was given exposures of as much as eight hours without a break. For days, in Dr. North's opinion, she lived on infra-red rays; when the lamp was there she revived, when it went out she became worse.

For all practical purposes the patient survived the pneumonia, but a purulent cystitis established itself, and she died eleven days after admission to hospital, the certificate stating that death was due to toxæmia following pneumonia and purulent cystitis.

In the last few months several patients in the hospital suffering from pneumonia have received obvious benefit from infra-red radiation, especially in the relief of pain and the easing of respiration. Naturally the inductotherm would be more efficient and could be applied without any disturbance of the patient, but unfortunately the hospital has not yet got one, and mine is not portable.

The medical mind in Australia does not seem to be slow to receive what may be advances in other lines of treatment. Adequate trial appears to have been given to the use of gold salts in the treatment of tuberculosis and of arthritis; olive oil emulsion has had its trial, although any physiological reason for expecting benefit from these measures is obscure. Yet electrotherapy, in which every form of treatment is built on a foundation of sound physiology, is largely ignored. The whole medical profession uses heat because it knows that heat means increased blood supply and that all repair of tissue demands that increased blood supply. Yet too large a proportion of the profession clings to old, inefficient forms of heating that do not penetrate, such as foments and hot air *et cetera*, when they should use the infra-red lamp, the inductotherm or diathermy.

Several years of experience, of which this paper is a short summary, convince me that it is reasonable to say that in Sydney, and probably in the other great Australian cities, many lives are lost and an infinite amount of avoidable suffering is permitted because of the inertia and the prejudice of many of the senior members of our profession.

Acknowledgements.

I wish to thank all the medical practitioners in this district for permission to publish the cases described in this paper.

Reports of Cases.

REMOVAL OF A PARIETAL ASTROCYTOMA.

By GILBERT PHILLIPS, M.S., M.Sc.,

Honorary Neurological Surgeon, Lewisham Hospital; Honorary Assistant Neurological Surgeon, Royal Prince Alfred Hospital.

(From the Department of Surgery, University of Sydney.)

THE case reported here is of considerable neurological interest owing to the almost complete recovery of deep sensibility in a patient the major portion of whose right parietal lobe was removed during the extirpation of a glioma.

History.

The patient, Mrs. M.C., aged twenty-seven years, was referred to the neuro-surgical unit at Lewisham Hospital on April 8, 1937, by Dr. John Herlihy. She had always been a healthy woman. There was no history of intracranial disease in the members of her family.

Her present illness commenced six years before, when she first suffered from convulsions, which were sudden in onset and took place originally at night, during sleep. The attacks were infrequent at first, and several months would often elapse between them. Similar attacks then began to appear in the day, without warning and with no auræ. Consciousness was lost in these attacks. The tongue was bitten; but there was no incontinence, and at that time there were no focal movements of the face or extremities. In the previous three years twelve attacks had occurred, each one preceded by twitching in the left arm and by a subjective sensation of heaviness and tightness in the left arm and left leg. These attacks were succeeded by severe headaches, which were general in distribution. In the previous twenty months she had a number of minor attacks, in which twitching in the left side of the face and the left arm and leg took place, but consciousness was not lost. In the previous twelve months she had suffered quite frequently from early morning headache. For two years she had noticed very gradual deterioration of vision and had worn glasses for that period. Recently she had observed that any sudden movement might produce transient blindness. She also complained of frequency of micturition (eight to ten times during the day and two to four in the night).

Physical Examination.

On examination on April 8, 1937, the patient appeared to be a well-nourished young woman who was well oriented and alert, and cooperated well in subjective tests. She was right-handed. The head was not enlarged, there was no neck rigidity, and no bruits were heard. There was no anosmia. The visual acuity was $^{6}/_{12}$ in each eye, and there were four diopters of papilloedema in each fundus. The fundal veins were distended. The ocular movements were full and the ocular reflexes were normal. Nystagmus, which was unsustained, appeared on right and left deviation of the eyes. There was no disturbance of facial sensation or power. When protruded, the tongue deviated to the left. Hearing was not affected, and Weber's test was not referred. In the upper extremities there was no disturbance of posture, power, reflex action or superficial sensation. There was a complete loss of passive position and passive movement sense in the terminal phalanges in the fingers of the left hand, but not at the left wrist, and there was considerable loss of tactile localization in the left hand and a relative disturbance of tactile discrimination in the same situation. Vibratory sense was normal and astereognosis was not present. No abnormal neurological signs could be detected in the lower extremities except a relative loss of passive position sense at the left great toe and tenderness on deep palpation of

ANAESTHESIA CHART

Name Mrs. M. C. Ward 10 Age 27 Date of Operation 9-4-37.

Lungs Clear Heart Clear

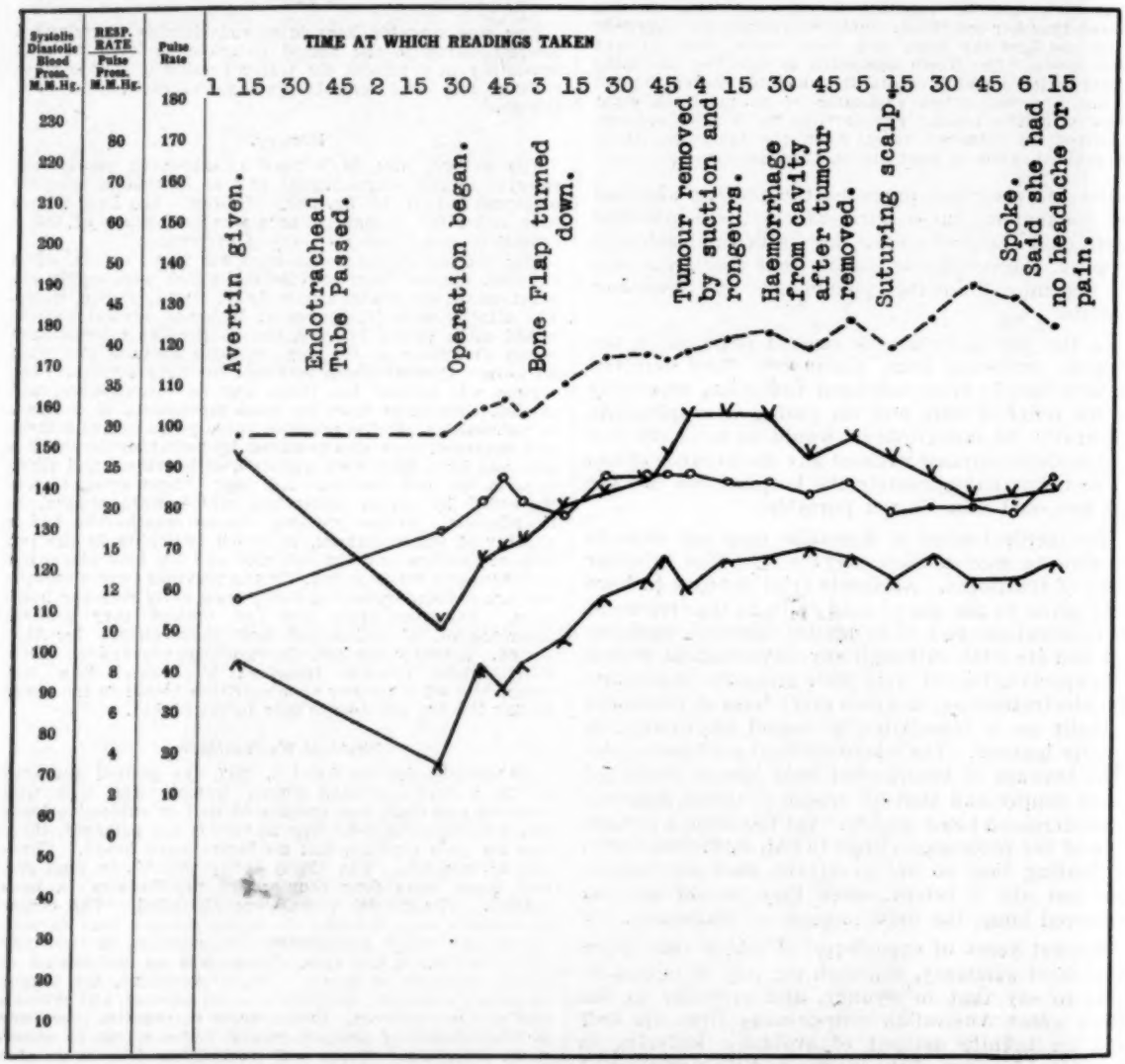
Urine N.A.D. Blood Pressure S150, D100 Rectal Temperature

Operator Gilbert Phillips 1st Assist F. Ellis Preliminary Drugs Atropine gr. $\frac{1}{100}$

Operation Craniotomy Anæsthetic Avertin and Gas 02 Amount 7.5c.c. in 312c.c.

Method of Administration

Time of starting anæsth., beginning of op., removal of anæsth., and close of op., to be recorded on chart.



CODE:—● PULSE RATE; O RESPIRATION RATE; V SYSTOLIC AND DIASTOLIC BLOOD PRESS.; X PULSE PRESS.

Induction :— Avertin P.R.; Gas 02 Endothracheal, Heidbrink Absorber Method.

Subsequent anæsthesia :— 50c.c. 1% Novocaine into scalp.

Condition on leaving operating room :— Excellent

Drains ---------- Anæsthetist H. J. Daly.

Chart showing the stages of the operation for removal of an astrocytoma of the parietal lobe.

the calf muscles of the same leg. Both plantar responses were flexor. The abdominal reflexes were present on each side. Her gait and station were normal. Bjerrum screen examination of the visual fields disclosed full fields in each eye with a three-millimetre test object at two metres. Diagnosis of right parietal tumour was made, probably meningioma, possibly a slowly growing glioma.

Operation.

At operation, after reflexion of a parietal bone flap, the dura was found to be very tense, and a brain needle was passed into the anterior horn of the right lateral ventricle. At a depth of seven centimetres a free flow of cerebrospinal fluid was encountered; but after removal of about fifteen cubic centimetres the tension was greatly reduced. The dura was then incised and elevated. A cyst, approximately the size of a hen's egg, was found on the surface of the parietal lobe, about one centimetre from the mid-line. It lay immediately behind the upper end of the right Rolandic vein. The cyst ruptured spontaneously, discharging about fifteen cubic centimetres of fluid. A solid tumour mass was then seen to underlie the cyst and to pass into the depths of the parietal lobe. This was removed piecemeal, partly by suction and partly by rongeurs, leaving a cavity immediately behind the Rolandic vein, about 1·5 centimetres lateral to the mid-line. The dimensions of the cavity at first were approximately six centimetres antero-posteriorly, five centimetres in the coronal plane, and about six centimetres in depth. During subsequent manipulation it greatly decreased in size to about five by four by four centimetres. Slight intractable hæmorrhage continued from the depths of this cavity, and a small muscle graft, which controlled the bleeding, was applied. The bone flap was wired back into position and the galea and scalp sutured with silk. The stages of the operation are recorded in Figure I. Histological examination of the tumour tissue showed it to be a protoplasmic astrocytoma (Figure II).

Subsequent History.

Convalescence was uneventful. All the sutures were removed by the fifth day and the patient was discharged from hospital, feeling perfectly well, on the twelfth day after operation. Three weeks after operation she was quite well and had had no headaches. Papilloedema had subsided to 1·5 diopters on each side. She was still complaining of a feeling of numbness in the left arm and leg. Examination of the visual fields disclosed no abnormality. The small decompression was soft. Examination of deep sensibility in the left hand and foot disclosed no disturbance at all of passive position and passive movement sense, no astereognosis and no disturbance of tactile localization; but the threshold for tactile discrimination on the back of the left hand was five centimetres, as against three centimetres on the back of the right hand. On the palm of each hand the threshold was two centimetres. Vibratory sense was intact. There was no loss of power in the left arm or left leg, and the abdominal reflexes were normal. There was an extensor plantar response of the left great toe.

Comment.

All modalities of deep sensibility, such as passive position and passive movement sense, vibratory sense, tactile localization and discrimination and stereognosis, are generally considered to be appreciated through the activity of neural mechanisms located in the parietal lobe of the brain. It is well known that all these forms of deep sensibility are not similarly affected by disease in this situation, and cases are frequently encountered of parietal lobe tumour or trauma in which one or more modalities of deep sensibility may be retained intact while the others are depressed or absent. The location of the lesion either at the surface or subcortically may play a part in these differential changes. For instance, it has been remarked that loss of vibratory sense appears only in subcortical lesions. The topographical site of the lesion, however, does not appear the only factor in the production of changes in deep sensibility. There seems little doubt that these forms of sensation may be appreciated beyond the limits of the parietal lobe. Charles Dowman,[1] for instance, has pointed out that disturbances of passive position and passive movement sense may occur after destruction of portion of the anterior central gyrus. In the light of the differential changes in deep sensation which may succeed parietal lobe disease it would appear not unlikely that some modalities may be appreciated in another situation as well, or that if the circumstances are favourable, if, for instance, the development of the lesion is slow, then neural mechanisms elsewhere are gradually educated to act as substitutes. Both these possibilities may be true, and a second one seems to be the only possible explanation of the neurological changes appearing in the course of the case described here. If in the slow destruction produced by the parietal astrocytoma some other mechanism was being integrated elsewhere for the appreciation of passive

FIGURE III.

Patient three weeks after operation.

position, passive movement, tactile localization and tactile discrimination, and was incompletely coordinated or was inhibited by the neural disorder resulting from the presence of the growth, an explanation would be available to interpret the reappearance of these forms of sensation after removal of the growth, even though the greater

FIGURE IV.
Operation site three weeks after operation.

portion of the parietal lobe had also been removed at the same time. It would be necessary to assume that vibratory sense and stereognosis are appreciated by a much more extensive mechanism or that the latter sense had developed at an early stage of the disease in some other situation.

Reference.

(1) Charles Dowman: "Kinesthetic Function of Precentral Convolution: Evidence Obtained by Alcohol Injection in Jacksonian Epilepsy", *Southern Medical Journal*, Volume XX, May, 1927, page 348.

Reviews.

SURGICAL PROCEDURES.

THE principle adopted in Volume I of the eighth edition of "The Operations of Surgery", by Rowlands and Turner, has been followed in Volume II, "The Abdomen",[1] many chapters having been rewritten and revised by specialists in various branches, thus making available the latest ideas and details of technique to the reader.

W. H. Ogilvie deals with the preparation of the patient and with operations on hernia, the spleen, the pancreas and the anus and rectum. A. Ralph Thompson deals with most of the urological sections, while G. F. Hibberd has undertaken the gynæcological portions and R. C. Brock is responsible for the section on thoracic surgery.

Many of the old procedures have been discarded; but the authors have retained some that are of historical interest only and could equally well be omitted in favour of some more intimate detail of generally accepted procedures. Mr. Ogilvie gives a practical if short résumé of post-operative pulmonary complications which should be of interest to all surgeons. His remark that the injection treatment of hernia is "a throw-back to mediæval methods" should deter anyone considering the employment of this method.

The late H. Barnard's classic on subphrenic abscess is epitomized and its essential details are clearly given. In view of E. S. J. King's recent article on gastrostomy this section should be reviewed in the next edition, as only the older methods are illustrated. Apart from this the section on surgery of the stomach is excellent and is made more valuable by the appearance in it of many first-class diagrams. The discussion on the treatment of appendicitis will be invaluable to all young surgeons and to many even of those experienced in such cases.

Diverticulitis, as merits its importance, is dealt with sufficiently to provide a sure groundwork for further experience to develop.

The article on hydatid of the liver is woefully inadequate and needs to be rewritten by a surgeon experienced in this condition. The comments that "any scolices which are within reach are next removed" and "if the cyst be crammed with scolices very little fluid escapes" will bring a smile to the face of any Australasian surgeon. The name of Harold Dew is mentioned; but it is obvious that his monograph on hydatid has not been consulted.

The chapter on surgery of the biliary apparatus is well written; but neither here nor in other sections is mention made of the value of a self-retaining retractor in abdominal work.

It is pleasing to see that mention is made of the operation for prostatectomy devised by the late S. Harry Harris. Some illustrations of the Harris operation are provided, but the main technique given is founded upon Freyer's original account; the value of lighted retractors in this work is not stressed, nor is the advisability of securing as complete hæmostasis as possible.

In view of the success that has attended the Wreden Stone technique for rectal prolapse, the inclusion of a description of this method would render the discussion on the operative treatment of this difficult condition more complete. The most effective procedures for excision of the rectum, including Devine's methods, are discussed. This section is an excellent one.

In a review of the methods of lumbar sympathectomy Royle's name is mentioned, but no detail of his technique is given, although it is preeminently the most convenient way of dealing with the sympathetic in cases of Hirschsprung's disease.

The final chapter deals with recent developments in general surgery and should prove most interesting and valuable reading for all practising surgeons.

This volume is a worthy companion to Volume I, and the printing, illustrations, bibliography and index are all first-class.

There should be no doubt as to this text-book's standing preeminent in the English language, and it maintains the excellent standard set by the original Jacobsen's Surgery. The joint authors are to be complimented on having carried out a difficult task very satisfactorily, as they have omitted all redundancy yet given essential details. The publishers deserve a meed of praise for their high-class work.

[1] "The Operations of Surgery. Volume II: The Abdomen", by R. P. Rowlands, M.S., F.R.C.S., and P. Turner, B.Sc., M.S., F.R.C.S.; Eighth Edition; 1937. London: J. and A. Churchill Limited. Medium 8vo, pp. 1007, with 514 illustrations, 4 of which are in colour. Price: 36s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 11, 1937.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE BIOLOGY OF SOCIAL LIFE.

AMONG the many orations and memorial lectures that are published from time to time in medical journals of the English-speaking world, there occasionally appears one of exceptional merit and of absorbing general interest. The seventeenth Maudsley Lecture on "The Biology of Social Life", delivered last November before the Royal Medico-Psychological Association by Sir Walter Langdon-Brown,[1] is one of these. Although this lecture is published in a medical journal and is written from the standpoint of a biologist, its arguments are so clear and it is so full of wisdom that it should be brought to the notice of all thinking men and women in the community.

The distinguished author begins by reminding his readers that man, being a social animal, is subject to the biological laws which govern life in general and gregarious animals in particular; yet this simple fact is generally forgotten. He shows, in short, how biological laws have operated in the past, and he makes it quite clear that if we cannot adapt ourselves to the demands of evolution, this civilization will go as others have gone before it. "The whole story of many-celled organisms is one of mutual adjustments, each trying to do the best for itself within the limits of those adjustments." In the first stage of evolution a number of single cells herded together for mutual support, but each did the same work; in the next stage groups of cells did different work. There are in evolution two parallel processes—an increasing division of labour and an increasing coordination between the different parts. Coordination has been achieved under the control of the central nervous system. To keep order, a strong central government is needed, and no high degree of differentiation is possible in the animal body without the control of a centralized nervous system, which has gradually acquired an increasing predominance. Sir Walter Langdon-Brown likens the origin of the central nervous system to a group of settlers on the coast, who gradually invade the interior, first singly and then in an organized army. Once established, the invader assumes control over the indigenous inhabitants, fortifying himself as he goes, and maintaining his protectorate by a system of rapid communication throughout the invaded areas. He points out that evolution of the vertebrate gave ample opportunity for the brain to expand. With the development of the higher centres, automatic actions of the lower centres were held in check and more skilled voluntary movements became possible; and as the highest level developed, it exercised control over both voluntary and automatic movements, restraining emotional expression, but increasing skill through increased intelligence. "Thus man, having laboriously acquired the power of speech, had to learn the still more subtle art of silence." The course of successful evolution has been to increase the size, not of the cell or of the individual, but of the unit—the unicellular became the multicellular; isolated individuals became a community. Sir Walter Langdon-Brown recalls the statement of Sir Arthur Keith that there are two opposing tendencies in life

[1] *The Journal of Mental Science*, January, 1937.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Meningitis Caused by Pfeiffer's Bacillus.

THE specific treatment of meningitis caused by Pfeiffer's bacillus is described by Le Roy D. Fothergill (*The New England Journal of Medicine*, April 8, 1937). The demonstration of various strains of the *Hæmophilus influenzæ* and the properties of the meningeal strain has made it possible to produce a specific immune serum. So far as is known, the immunological mechanism involved in the serum destruction of *Hæmophilus influenzæ* is a complemental bacteriolysis. Antibody has no destructive effect in the absence of complement. In only occasional cases of meningitis is complement demonstrable in the cerebro-spinal fluid. Antiserum has been produced by immunization of horses with virulent meningeal strains of the organism. The method of treatment of patients is as follows. Serum without complement is given intravenously once a day for the first two days in dosages of 30 cubic centimetres for an infant and 30 to 50 cubic centimetres for older children. This process is for the purpose of overcoming the bacteriæmia that is usually present. It is of little or no value in treating the infection in the subarachnoid space, as the meningo-vascular barrier has a very slight permeability for antibodies. A mixture of antiserum and complement is given intrathecally for as long as is indicated. This mixture consists of two parts of antiserum and one part of complement, the usual dose being 15 cubic centimetres of the former and 8 cubic centimetres of the latter. The cerebro-spinal fluid is completely drained before the mixture is allowed to run in by gravity, the amount run in always being less than the amount of fluid withdrawn. After each intrathecal administration the foot of the bed should be elevated, and the patient's position should be changed frequently during the next hour to facilitate the distribution of serum throughout the subarachnoid space. Such treatment is usually administered by lumbar puncture, although the cisternal route is frequently employed. One or two doses may be given to infants into the lateral ventricles through the open anterior fontanelle as a routine. Freshly obtained adult human serum is used as complement. Treatment should be continued longer than at first appears necessary, as periods of improvement may be followed by fatal relapses. Daily examination of the cerebro-spinal fluid will give the most reliable evidence indicating the course of the disease and its response to treatment. A fall in the total cell count, a return of sugar to the normal amount, and an increase in the percentage of mononuclear cells are evidence of improvement. Daily cultures of the fluid are the best guide. Continued fever despite clearing and sterility of the cerebro-spinal fluid is of grave prognostic omen. This form of treatment has apparently been associated with some reduction in the mortality rate even though the results are not striking. The author suggests that continued use of this therapy is indicated, since no other has as yet been successful.

Vitamin Therapy.

R. A. PETERS (*The Practitioner*, April, 1937) gives guiding principles for the use of vitamins in therapeutics and attempts to apply these in practice. The only object of separate dosing with vitamins is to raise the daily ration to the minimum amount required. Provided that this is reached, together with a reasonable reserve store, no further dosing with vitamins should do good. The vitamin content of good food may vary with cooking and storage; it is wise, therefore, to give a margin of vitamins to cope with fluctuations in the diet. In the case of the water-soluble factors of the *B* class vitamins and vitamin *C*, there seems to be no danger of overdosage, because excess is likely to be excreted in the urine. With the fat-soluble vitamins there is a possibility of over-dosage, but it seldom occurs. The average amount of each vitamin needed can be determined approximately for the individual; but there may be cases of marked idiosyncrasy. Not unrelated to this is the uncertainty that vitamins when given by the mouth are properly absorbed or destroyed in the intestine; if there is failure of absorption, a patient may be suffering from vitamin deficiency while vitamins are being administered in large amounts. Certainty cannot be reached until there are accurate and sufficiently simple methods of estimating the amount of vitamins used by the individual. Lack of symptoms at any given time does not necessarily indicate that a person's diet is correct, because the fat-soluble vitamins are stored for long periods. If a deficiency shows itself in any definite form, then it really means that the dietary fault has been in existence for some time. It has been recommended as a guide by the Health Section of the League of Nations that women during pregnancy and lactation require 8,700 of vitamin *B*, 1,120 of vitamin *C* and 340 of vitamin *D*. Rather less is necessary for the ordinary person, but children require the same quantity. The conditions that may require vitamin therapy are diverse. If the vitamin lack is acute, adequate therapy should induce a rapid change; on the other hand, if a person is suffering from a chronic lack of vitamin, there may be a long delay in improvement. If vitamin deficiency is suspected, treatment with the appropriate vitamin should be instituted; in most cases a definite improvement should be noticeable within two to four weeks. If there is no improvement, it must be assumed that the condition is not due to vitamin deficiency, or, alternatively, that the vitamin is not being absorbed. Injection may then be tried; if after a reasonable time there is still no change, there is no object in continuing the treatment. The question of idiosynerasy must not be forgotten.

Lobar Pneumonia.

F. L. HORSFALL, K. GOODNER, C. M. MACLEOD AND A. H. HARRIS (*Journal of the American Medical Association*, May 1, 1937) describe antipneumococcus rabbit serum as a therapeutic agent in lobar pneumonia. Antipneumococcus horse serum rarely possesses high mouse titre, and in order to obtain high potency concentration of the serum and increased expense are the rule. Horse serum sometimes causes anaphylactic reactions and chills, and usually causes serum sickness. It does not reduce the incidence of empyema. In view of these disadvantages unconcentrated antipneumococcus rabbit serum was used in the treatment of twenty-two patients with lobar pneumonia. All patients were tested for sensitivity to rabbit serum in a routine way by three methods: intradermal injection, conjunctival instillation and intravenous injection. If there was no reaction, two cubic centimetres of rabbit serum diluted to twenty cubic centimetres with saline solution were given, and the volume of serum was doubled every two hours until from thirty to forty cubic centimetres of undiluted serum were given at each injection; later it was found that the dose of serum could be increased more rapidly and given every forty minutes; in the last few cases one large dose (125 cubic centimetres) of undiluted serum was given in ten minutes in an attempt to produce an adequate therapeutic response. A chill occurred after some of the early injections; then it was found that heating the serum to 56° C. for thirty minutes and adsorbing with sterile kaolin reduced the tendency to chill, as did administration of 0·9 gramme of acetylsalicylic acid. Ten patients with type I pneumococcus pneumonia were treated with unconcentrated type I antipneumococcus rabbit serum. Serum was given on an average forty-two hours after the onset of pneumonia. All ten recovered. In one case a turbid pleural exudate full of type I pneumococci resolved after the intravenous administration of serum. Four type II patients infected with type II pneumococcus were treated with rabbit serum, the first dose being given, on an average, seventy-two hours after the onset of pneumonia. Two patients had consolidation of two lobes on

admission to hospital, and in two there were grossly infected pleural exudates. One of these patients died. Three patients infected with type VII and five with type VIII pneumococcus were treated with types VII and VIII pneumococcus antiserum; all recovered. Convalescence was uncomplicated except for serum sickness in three cases. In almost every case in which rabbit serum was used the temperature and the pulse rate and respiration rate fell to normal a few hours after serum was given.

Carcinoma of the Thyreoid in Children.

HUGH F. HARE (*Journal of Radiology*, February, 1937) gives a detailed classification of carcinoma of the thyreoid in children and discusses six cases. He considers that preoperative diagnosis in early cases is practically impossible, so that innocent adenomata must often be removed in order to protect the individual who has or who may later develop cancer. Treatment of all tumours of the thyreoid is primarily surgical, but in addition irradiation is indicated in all cases. Five of the six children under consideration are living and well today. All were under thirteen years of age. Growth has continued normally in all of these children following treatment and in none of these children has myxœdema followed treatment. Two of the six cases occurred in lateral aberrant thyreoid tissue and one occurred in a thyreoglossal cyst. Six cases of cancer of the thyreoid occurring in children under thirteen years of age are reported; five of the children are today living and well.

NEUROLOGY AND PSYCHIATRY.

Polioencephalomyelitis due to Botulism.

GABRIEL A. SCHWARZ (*The Journal of Nervous and Mental Disease*, July, 1937) reports a case of polioencephalomyelitis due to botulism. The patient was a girl, aged sixteen years. The illness commenced with nausea and epigastric discomfort. These were quickly followed by the onset of bilateral ptosis and photophobia. Double vision occurred together with difficulty in swallowing. Two days later she experienced difficulty in walking and inability to hold things in her hands. Weakness became more prominent. Dizziness was constant. Four days after the onset of the illness the patient became restless and had incontinence of urine. Death from respiratory failure ensued on the fifth day of the illness. The gross and microscopic pathological findings are recorded. The changes in the central nervous system corresponded with those previously described in such cases. Pronounced œdema of the small and middle-sized pyramidal cells of the third lamina of the agranular cortex and a reduction in the number of small cells in the red nucleus were features that had not been reported in other cases. The author, after discussing botulism and the exotoxin of *Bacillus botulinus*, expresses the opinion that the pathological changes in the central nervous system in his case did not fully explain the neuro-paralytic phenomena observed clinically.

Reaction of Certain Psychotic Types to Alcohol.

CARL E. TRAPP AND PURCELL G. SCHUBE (*The Journal of Nervous and Mental Diseases*, June, 1937) describe a method whereby they believe it is possible to investigate the mental capacity of certain types of psychotic individuals by the use of alcohol. The method consists in giving patients a liquid containing 40% of alcohol through a nasal tube. An initial dose of 30 cubic centimetres, followed by an equal quantity of water, is given. Following this, every four minutes doses of 15 cubic centimetres are given, to a maximum of 120 cubic centimetres; then 15 cubic centimetres are given every ten minutes, to a final maximum of 180 cubic centimetres. During this procedure the patient is observed and questioned. To check results certain patients receive a repetition of the treatment. The authors' patients included persons with various forms of *dementia præcox*, psychotic mentally defective persons, and some with manic-depressive insanity. Histories of these patients are given in detail. The authors believe that by this method it is possible to diminish temporarily certain types of mutism and to distinguish between the stupors of *dementia præcox* and manic-depressive psychoses. They also believe that they are enabled to probe the intellectual level underlying certain depressive states; but simple *dementia præcox* and certain types of mental deficiency could not be differentiated, and no permanent therapeutic benefit was noticed in any of the patients so treated. The paper contains a discussion upon the therapeutic uses of alcohol.

Gonadal Disturbances in Behaviour Problems.

MATTHEW MOLITCH AND SAM POLIAKOFF (*The American Journal of Orthopsychiatry*, October, 1936) studied the behaviour problems in eighty-one boys with hypergonadism, hypogonadism, cryptorchidism or undescended testicle, and delayed secondary sexual development. The patients were studied and grouped according to diagnosis, mental level, school achievement, behaviour, personality and treatment. The authors find that except for delayed secondary sexual development, gonadal disturbances are not of frequent occurrence in boys with behaviour problems. Boys with hypogonadism tend to be brighter than those with hypergonadism, the latter being below the average. In the authors' series, boys with undescended testicles presented the most serious school behaviour problems. Sexual offences were quite rare, one boy only, with hypergonadism, being guilty. The boys generally with gonadal dysfunctions were found to be unstable, immature and infantile, the children with cryptorchidism the more so. The authors claim excellent results from the treatment by an anterior pituitary-like substance ("Follutein") in seven out of eleven cases of cryptorchidism. They recommend this treatment within the first few years of life before any surgical intervention. Children with delayed secondary sexual characteristics were benefited by the use of growth hormone, which also contains some sex hormone. Treatment of hypergonadism at present appears unsatisfactory.

"Musicogenic Epilepsy."

MACDONALD CRITCHLEY (*Brain*, Volume LX, Part I, 1937) quotes in detail a number of cases of what he calls "musicogenic epilepsy" in contradistinction to "acousticomotor epilepsy". In acousticomotor epilepsy the fit is induced by sudden and unexpected noise, whereas in "musicogenic epilepsy" it is induced only by music. The fit in "musicogenic epilepsy" may vary from transient obfuscations of consciousness to complete major attacks, with convulsions and incontinence. Some attacks do not follow closely on the hearing of music, the stimulus having to be maintained for many minutes before it occurs. Ten such cases are discussed in detail, and the literature is referred to for similar cases which have been described by Merzheevsky, Trutovsky, Oppenheim and others. The pathogenesis of this type of epilepsy is discussed, particulary in regard to the similarity to hysteria, the author emphasizing the danger of drawing a false distinction between "organic" and "functional" epilepsy.

Economic Security and Children's Attitudes to Parents.

H. HELTZER (*The American Journal of Orthopsychiatry*, October, 1936) investigates the nature and extent of the relationship that exists between the economic level and the children's attitudes to parents, and concludes that economic insecurity makes for emotional insecurity. Generally the children from the lowest economic level compare unfavourably with others. On the other hand mere economic security does not necessarily imply emotional security. The "problem" child is not the special privilege of any one class, and this study shows that the most satisfactory emotional balance and security are found in children of the middle classes. The author looks to social reconstruction to minimize the economic frustrations of the lower classes.

British Medical Association News.

SCIENTIFIC.

A SPECIAL MEETING of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, East Melbourne, on Thursday, July 29, 1937, PROFESSOR R. MARSHALL ALLAN, the President, in the chair.

Poliomyelitis.

DR. MOSTYN L. POWELL read a paper entitled "Poliomyelitis" (see page 419).

DR. REGINALD WEBSTER said that those present had heard an excellent presentation of the clinical features of poliomyelitis, and to him had been allotted the task of discussing the pathology of the disease. He would endeavour to do so in as concise a manner as possible by demonstrating lantern slides showing the morbid histology of the disease as he had been enabled to study it in the course of his work at the Children's Hospital.

In a presentation of the pathology of any disease condition, pathogenesis demanded consideration at least equal to that given to morbid anatomy, and he therefore proposed to include some discussion of the mode of infection and the method of spread of infection within the body.

He said that to affix a high-sounding title, such as "The Micropathology of Poliomyelitis", to this demonstration would possibly create apprehension in some minds of an excursion into the intricacies of neuro-pathology; but the sequence of events in poliomyelitis was not at all complex. It was nothing more nor less than the working out of the fundamental process of inflammation, which retained its essential unity in whatever tissue or organ it was initiated.

Dr. Webster said that the majority of the photomicrographs that he was about to show had been prepared from sections of the cervical part of the spinal cord and medulla of two children who had died from poliomyelitis early in the course of the present visitation; but in order to give a more complete review of the micro-pathology the slides would be supplemented with others obtained from experimental poliomyelitis that he had induced in monkeys with material obtained during a former epidemic.

In the present epidemic there had been an unhappy predominance of bulbar symptoms; but the lesions inflicted by the virus might be found anywhere or everywhere in the cord, from the sacral region to the mid-brain. They rarely extended higher. A "cerebral" type had been described; but the evidence in favour of the condition's being poliomyelitis was purely clinical. The cerebrum did not appear to be susceptible to the poliomyelitis virus, and in this connexion it was interesting to note that when a monkey was inoculated with the virus intracerebrally the lesions occurred not in the cerebrum, but in the brain-stem and cord.

The several types of lesion he proposed to show might be tabulated as follows: (i) Perivascular, (ii) diffuse interstitial inflammatory reaction, (iii) focal interstitial inflammatory reaction, (iv) all degrees of damage to nerve cells, (v) changes in the posterior root ganglia, (vi) meningeal reaction.

By the time such table was exhausted it would no doubt be appreciated that the virus attacked on a wide front, with vital strategic points as its immediate objective.

Perivascular Cell Collarettes.

Dr. Webster said that the perivascular cell collarettes (Figure I) were perhaps the most striking and obvious histological features in a section of spinal cord damaged by and reacting to the virus of poliomyelitis; but the degree of prominence they assumed was not a measure of their diagnostic significance. They were merely the expression of a non-specific inflammatory reaction in the central nervous system and were just as much a feature of epidemic encephalitis, rabies and post-vaccinal encephalitis as of poliomyelitis. They were not even distinctive of virus diseases, but were to be observed in the brain in general paralysis and other forms of cerebro-spinal syphilis, and in chorea and in the neighbourhood of a cerebral abscess.

Dr. Webster next showed a slide depicting the aggregated inflammatory cells distending the vascular adventitial spaces and appearing to dissect the adventitia of the vessel into its component layers. The cells were mainly lymphocytes, with a small proportion of polymorphonuclear leucocytes and plasma cells. Dr. Webster said that these cells were frequently confined to the adventitial spaces, but might overflow into the perivascular lymph spaces, and although their origin had been a subject of controversy, they were most probably derived from the circulating blood. In this, as in other virus diseases, the perivascular lymph space was often curiously dilated, although not necessarily occupied by cells. Dr. Webster said that congestion was the rule and hæmorrhages were frequent.

The next slide shown by Dr. Webster revealed the extreme to which interstitial hæmorrhage might be carried. The anterior cornua of the spinal grey matter were delineated by gross hæmorrhage of a degree sufficient to permit of a direct macroscopic photograph. The specimen had been obtained from a child who had died during a former epidemic of poliomyelitis, and no staining or other artifice had been employed to heighten the effect. Dr. Webster said that he should not like to leave the impression that this was a common occurrence; it was quite exceptional.

The next slide showed a macroscopic view of the lumbar part of the cord, in which there was gross hæmorrhage in the anterior cornua.

Interstitial Lesions.

Dr. Webster then showed slides depicting changes, which, he said, though much less striking than the cell collars at first glance, were really more characteristic of the disease. Inflammatory cell reaction appeared in two forms: (i) a diffuse infiltration, (ii) defined focal collections. The focal collections were of special significance, for they were never seen in epidemic encephalitis. Many of the cells might be polymorphonuclear cells—another distinguishing feature as between poliomyelitis and encephalitis.

By ordinary staining the inflammatory cells appeared to be of lymphoid type, with a varying admixture of polymorphonuclear cells; but by the silver carbonate method of staining the bulk of the diffuse and focal exudate was found to consist of microglia, the "Hortega" cells, which were now regarded as the essential scavengers of the central nervous system. The microglia would come up for further discussion in the consideration of the process of neuronophagia at a later stage.

Nerve Cells.

Examination of the nerve cells invariably showed degenerative changes. In experimental poliomyelitis such changes were to be observed even in animals killed in the preparalytic stage. The most severe lesions were in the anterior cornua; but the cells of the posterior horn and those of Clarke's column were also attacked (Figure I).

Dr. Webster said that the changes in the nerve cells consisted of poor staining and loss of Nissl granules, eccentricity of the nucleus, and swelling of the axis cylinder, and were frequently so severe as to culminate in total necrosis and complete disappearance of the cell. The dead cells became surrounded and invaded by phagocytes, a process known as neuronophagia. Dr. Webster showed a slide made from a low-power photomicrograph, in which the nerve cells in the anterior horn were seen to have a dotted appearance owing to the occupancy of neurophages. A high-power view of the same section showed one intact ganglion cell, one degenerate with loss of nucleus and poor staining, and one invaded by neurophage cells.

Dr. Webster went on to say that the process of neuronophagia was effected mainly by those derivatives of the microglia known as "Hortega" cells, although polymorphonuclear cells also played a part. The microglia

cell was both amœboid and phagocytic and the view had been advanced that the microglia was but another unit in the far-flung defensive line of the reticulo-endothelial system. In the discharge of its scavenging function the microglia cell underwent a transformation into what was known as a compound granular corpuscle, and in appropriate preparations such corpuscles might be seen lying along the blood vessels, distended with myelin, and apparently about to discharge their contents into the vessel. The process of cell death might be very rapid. In the study of experimental poliomyelitis it had been found that twenty-four hours made all the difference between early lesions in the nerve cells and their conversion into a necrotic mass of *débris*. The significant observation had been made that the intensity of the changes in the nerve cells bore no relation to the vascular lesions and there could be little doubt that the death of the cells was not a secondary effect of the vascular lesions, but was due to a direct attack of the virus upon them.

Posterior Root Ganglion.

Dr. Webster said that lesions in the posterior root ganglia were very common. They were essentially the same as those occurring in the spinal grey matter—degeneration and necrosis of the ganglion cells, collections of inflammatory cells and neuronophagia. Perhaps it was not generally appreciated that the posterior nerve roots also showed inflammatory reaction. He showed a slide (Figure V) in which the appearances supplied an anatomical basis for the pain and hyperæsthesia that were frequently such distressing features of poliomyelitis. Dr. Webster said that it seemed remarkable, in view of the striking and unequivocal character of the lesions in the posterior root ganglion, that so little objective sensory loss could be demonstrated clinically. The suggestion had been made that the spine sign was to be referred to traction on the inflamed roots and ganglia. The lesions in the posterior root ganglion were histologically identical with those occurring in *herpes zoster*, which condition had been described as the sensory analogue of poliomyelitis.

Dr. Webster then showed a slide prepared from a photomicrograph of the posterior root ganglion in experimental poliomyelitis (Figure V) and one from a photomicrograph of the posterior root ganglion in human poliomyelitis. He remarked that the similarity between the two slides was such that one could hardly be distinguished from the other, and the faithful reproduction of the disease in the monkey shown in the ganglionic lesions was seen throughout the spinal cord and brain stem.

Meningeal Reaction.

Dr. Webster said that the degree of inflammatory reaction in the meninges was a very variable quantity. Such reaction was not always demonstrable even when the patient (or laboratory animal) had exhibited those particular manifestations of irritability that were commonly ascribed to meningeal irritation. In the case of the boy from whose tissues the next photomicrograph had been obtained, Dr. Webster had made particular inquiry as to whether the stiff neck and spine sign had been present. He had been assured that they had been; but in section of the cervical cord shown the only inflammatory reaction in the meninges that he could detect was one small focus in the pia-arachnoid where it dipped into the antero-lateral fissure of the cord. Dr. Webster said that in the photomicrograph the meningitis might appear to be considerable; but the high magnification tended to exaggerate it. It was the only focus to be found after examination of the whole periphery of the cord in a section in which the intramedullary lesions were as severe as any he had demonstrated that evening. On the other hand, meningeal inflammatory reaction might be conspicuous, as was shown in the next photomicrograph, obtained from the cord of a monkey (Figure VI).

It was customary to attribute the high cell counts in the cerebro-spinal fluid in the initial stages of the disease to an early meningeal lesion; but an inspection of the first slide (Figure I) would show a possibility of entry of cells to the cerebro-spinal fluid quite independent of meningeal inflammation. The cells could be seen distending the vascular adventitial spaces. Should they spill over into the perivascular lymph space they gained access to the cerebro-spinal fluid. The perivascular lymph space was formed by a prolongation of the pia-arachnoid along the sheaths of the vessels entering the cord; a communication had been demonstrated between the perivascular lymph spaces and the Virchow-Robin spaces in the adventitia. In general poliomyelitis was a satisfactory disease for the correlation of symptoms with lesions; but it became less so when the meninges failed to show the anticipated reaction in the presence of well-marked meningism. Hurst (*The Journal of Pathology and Bacteriology*, Volume XXXII, 1929, page 457) found that in the preparalytic stage of the disease in the monkey, when the only symptoms were those of irritation, there was no meningitis, although even at this early stage the motor cells in the anterior horn had begun to show signs of degeneration.

Dr. Webster said that, having reviewed in detail the microscopic lesions in poliomyelitis, he felt bound to comment that very few of them were exclusive features or had any claim to be regarded as specific for the disease. Many of them were common to all virus diseases of the central nervous system; but in respect to poliomyelitis it might be said that the lesions were more intensely focal and more directly destructive of nerve cells than in other virus infections.

Before concluding, he thought he might be permitted a few observations on the method of spread of infection within the body. That the portal of infection was olfactory mucous membrane was generally accepted. The conception of the method of spread that most entertained was that a phase of systemic infection supervened, which might not be followed by localization of the virus in the central nervous system and particularly in the spinal cord. In the vast majority of infected individuals such localization was not effected and the disease was "abortive". This hypothesis, which had for so long adequately explained the clinical phenomena, had recently been challenged, and the view had been advanced that the virus, being inherently and strictly neurotropic, was incapable of inducing a systemic infection, and that it spread from the olfactory mucous membrane directly along the axis cylinders of the olfactory filaments to the olfactory bulb. From the olfactory bulb the virus spread along the olfactory tract to the thalamus, involvement of which was held to be responsible for the constitutional symptoms early in the disease. Thence the path was, still by the axis cylinders, via the spino-thalamic tract, to the posterior horns of the cord and posterior root ganglia, and finally the anterior horns. Symptoms were thus first sensory and later motor.

Experimental evidence was adduced that the virus spread by way of the axons and not by the blood or cerebro-spinal fluid, it having been shown that when the cord was inoculated at various levels and the segments isolated by cutting the cord above and below there was no spread of infection.

If the olfactory nerves of an animal were cut and the virus was then introduced into the nasopharynx, the nervous system would not be involved.

Strong support was given to the doctrine of axonal spread by the work of Goodpasture (*American Journal of Pathology*, Volume I, 1925, page 29) on encephalitis. This worker, using the virus of *herpes simplex*, showed that the route followed by the virus was along the axis cylinders of the nerves supplying the part whether sensory, motor or sympathetic, that it was kept in place by the myelin sheath, and that it spread out on reaching the centre from which the nerve arose. When the masseter muscle was inoculated with the virus the initial lesion was found in the motor nucleus of the fifth cranial nerve in the pons; inoculation of the cornea resulted in an initial lesion in the sensory root of the fifth nerve in pons and medulla; when the hind leg was inoculated there was an acute myelitis in the lumbar region of the cord. From the

initial site the disease rapidly became diffused so as to involve the whole brain and cord. Although these results had been obtained with the virus of herpes, similar results with the virus of poliomyelitis had been obtained previously by Flexner and Lewis and by Landsteiner and Levaditi.

Dr. Webster said that possibly most people would be reluctant to abandon the conception that had served so well, that of a systemic phase, succeeded by invasion of the central nervous system by way of the blood and cerebro-spinal fluid, in favour of the doctrine of axonal spread. One fact, however, had always been a difficulty to him in the way of full acceptance of the systemic idea, and that was that the virus had never been demonstrated in the cerebro-spinal fluid. The theory of axonal spread disposed of this difficulty; but he was not sure that other difficulties did not arise. For instance, what was to be said about the abortive case?

The experimental basis for the idea of axonal spread was very attractive; but the ever-present need for care in arguing from the experimental animal to the human subject impelled him to keep an open mind on the question for the present. Also the close contact with the clinical world which the work of a hospital pathologist entailed had taught him to give due weight to clinical evidence and had on more than one occasion supplied a healthy corrective to the natural tendency of the laboratory worker to test-tube dogmatism.

DR. F. M. BURNET said that his contribution to the discussion could be concerned only with the more theoretical and experimental questions in regard to poliomyelitis. He would try to be as relevant as possible by touching only on those aspects of experimental and epidemiological work that had a direct bearing on the problems of prevention and treatment.

Owing to technical difficulties in isolating the virus there was little direct knowledge about the details of the process of infection in children. The current interpretation of this process was based on data from (i) the study of the experimental disease in monkeys, (ii) the epidemiological characteristics of the disease, (iii) the distribution of antibody in human populations, and (iv) the observed effect of certain prophylactic and therapeutic procedures applied to exposed populations or patients.

From such data a consistent and reasonable picture of the process of infection had been built up, and it was difficult to conceive that its outlines were seriously wrong. But it was not derived from direct study of the human infection itself, and there must always be a possibility of some serious undetected flaw in any indirect reasoning of this sort. With this reservation he proposed to summarize, rather dogmatically he thought, the present point of view of most virus research workers and epidemiologists.

He said that poliomyelitis was an infection due to a filtrable virus—the smallest virus known, only two or three times the diameter of a protein molecule. The virus, like all other viruses, could multiply only within living cells of suitable type. Active and repeated study had shown that only cells of the nervous system of man and certain species of monkey could allow multiplication of the poliomyelitis virus. A recent important experiment showed that while the virus would grow well in tissue culture of embryo human brain, it would not grow in any of the other types of embryonic human cell that were tested; nor would it grow in embryonic chick cells, which would allow the growth of nearly all other viruses not strictly limited to cells of the nervous system. Dr. Burnet wished particularly to stress this absolute neurotropism of the virus as the most important point in determining the characteristics of the infection. If it could be shown that poliomyelitis was not always neurotropic, a very radical modification of current theories would be necessary.

Dr. Burnet's next point was that epidemiological and serological evidence indicated that in almost every human community the virus of poliomyelitis was as ubiquitous as the diphtheria bacillus or the influenza virus. Parts of New Guinea and certain other Pacific islands were the only places in which epidemics with the characteristics of virgin soil epidemics had occurred. The virus must be constantly passing from person to person in the community, producing as its standard effect an inapparent or subclinical infection in the upper part of the respiratory tract of persons not previously immunized by such an infection. If the assumption of the obligatory neurotropism of the virus was correct, the commonest site of primary infection must be the olfactory mucosa, the only site at which free nerve fibrils were exposed to the environment.

Combined clinical and epidemiological evidence suggested that infections by the poliomyelitis virus might be of three types: (i) completely subclinical, with no signs referable to the central nervous system, infection being limited to the olfactory mucosa; (ii) non-paralytic, but showing definite evidence of involvement of the central nervous system (here there was a spread through the olfactory tract, limited at some point before the spinal cord was reached); (iii) paralytic infections in which the virus spread to the anterior horn cells of the spinal cord. There was no evidence of spread of the virus in the body by other than nervous pathways. Non-paralytic infections of the first and second types produced circulating antibody and induced active immunity.

Immunity to poliomyelitis was not apparently a simple humoral immunity due to circulating antibody. Monkeys might be inoculated subcutaneously with virus and develop circulating antibody; but they remained susceptible to infection by intranasal instillation of the virus. The failure to influence the disease in children by the administration of immune serum therapeutically and the recent failure of prophylactic serum inoculations in the Los Angeles epidemic would indicate that the relative unimportance of circulating antibody held also for human beings. Once a monkey survived a symptomatic infection with poliomyelitis it was immune to intranasal instillation of virus. It seemed that antibody, to be effective, must be produced in or near the susceptible cells. The same probably held for children.

Dr. Burnet said that, if this bald summary was correct, the opportunities of doing anything useful to prevent or treat the acute stages of poliomyelitis were limited. In a disease that normally produced a high proportion of subclinical immunizing infections, the rational mode of approach toward its prevention was to imitate such subclinical infections by some type of vaccination. Theoretically only intranasal inoculation with an attenuated virus could offer any real hope of success. The extreme danger of human experiments to that end was likely to prevent its ever being attempted. Temporary prophylaxis by inoculation of immune serum or whole blood had failed at Los Angeles, and appeared to have little theoretical justification.

Treatment in early preparalytic cases by immune serum had no theoretical justification if the virus was strictly neurotropic, and since reports of all controlled series so far published had shown that no clearly demonstrable benefit was obtained from its use in practice, the facts seemed to fit in with theory. Dr. Burnet said that it seemed to him that the only useful method of attack was to try to determine what were the reasons why one child developed paralysis while 99 others also infected had only mild non-paralytic symptoms or no symptoms at all. He felt that perhaps the present epidemic might provide a very useful clue in this direction.

There seemed to be no escaping the fact that in the early stages of the present outbreak it had been unusually easy to trace infection from case to case. In other words, an unusually high proportion of infections had given rise to clinical symptoms. Possibly a detailed analysis of these first cases would provide the clue to the greatest difficulty in the interpretation of the epidemiology of poliomyelitis. Very tentatively one might suggest that the factors requiring special consideration were the distinctly unusual weather conditions during June and early July, and the associated conditions in regard to the prevalence of colds and nondescript respiratory infections in the infected areas. As a working hypothesis that might be worth the attention of those directly concerned with the outbreak, he could imagine that the most highly infective

contact was that between a child who had a nasal infection with poliomyelitis *plus* a common cold, being thus apt to spray relatively large doses of virus into the air in coughing *et cetera*, and, as a recipient, a child who had been free from any cold for some time and had quite unobstructed nasal passages.

The simplest conceivable explanation of the patchy incidence of paralytic poliomyelitis was that the deposition of a relatively large dose of virus on the olfactory mucosa was the determining factor. Minute doses would be assumed to produce merely immunizing subclinical infections.

Dr. Burnet said that the only merit he could claim for the hypothesis was that it was easily susceptible to direct clinical test. He would be very interested to hear, for instance, whether mouth-breathers were relatively less affected than those with clear nasal passages, and whether paralytic infection was ever noticed in a child who had had a cold and running nose for the previous fortnight. In brief, all that was required to substantiate or disprove the hypothesis was careful clinical observation and record.

DR. H. N. FEATONBY said that, from the point of view of public health administration, the main points concerning poliomyelitis were that the infective agent was a virus and that the mode of entry was by the naso-pharynx. The infection was spread by direct contact with the naso-pharyngeal secretions of affected persons or temporary carriers. Infantile paralysis was therefore one of that large group of diseases, including diphtheria, scarlet fever, measles, mumps and whooping cough, that were spread by similar means. Preventive measures in all these diseases were therefore based on similar principles. Control was admittedly difficult, as the interchange of nasal secretions, especially between children, was on a large scale. The major triumphs of preventive medicine had been in relation to diseases with other modes of spread. For example, typhoid fever had been largely eliminated by the installation of sewerage systems and the provision of safe water and milk supplies; and in such diseases as malaria and plague, measures for destroying the insect vector might be employed.

The time-honoured methods of control of diseases spread by contact were: (i) isolation of patients, (ii) isolation of contacts, (iii) disinfection of persons and things.

With the discovery that persons, while showing no signs of a disease, might carry in the nose and throat pathogenic organisms and so be capable of infecting others, terminal disinfection had been relegated to a very unimportant place as a preventive measure.

Dr. Featonby said that, in passing, it might be useful to state that substances of the phenol group were rather ineffective against the virus of poliomyelitis and that disinfectants that are oxidizing agents, such as hydrogen peroxide, permanganate of potash, or the chlorine group, were the most efficient. Ultra-violet radiation was said to kill the virus with great rapidity.

Tests had been developed to detect individual susceptibility to diphtheria and scarlet fever, and methods of artificial immunization against them were now available. Unfortunately in poliomyelitis there was no test for susceptibility nor any method of artificial immunization, though research on these matters was being very actively pursued.

The features usually characterizing epidemics of poliomyelitis were that isolated cases occurred with no possible contact with known cases; rural communities were affected as much as or more than urban; the highest incidence was in children, and there was no relation with poverty, overcrowding or the nutritional state of the victim. If cases occurred in institutions, such as orphanages, only a small minority of the inmates acquired the disease in a severe form, and multiple cases of actual paralysis in families were the exception rather than the rule. The only epidemiological theory that would accord with these facts was that the virus was widespread throughout the community and that only a few individuals among the exposed acquired the infection, and that a large number of people in times of epidemic were temporary carriers. The truth of this theory had been strengthened by recent accretions of knowledge. It had been shown that the blood of most adults in urban communities contained antibodies to the virus, thus proving that most people had been exposed to the infection and most were immune, and that the high incidence of the disease in children was due to the fact that large numbers of them had not yet acquired immunity.

When the infection had been introduced to a Pacific island, where no previous contact with the virus had been established, persons of all age groups had been equally affected.

These known epidemiological facts showed why the closure of schools and all quarantine measures had been failures, whenever and wherever tried.

The measures recommended by the consultative council in the present epidemic had been put forward because this epidemic had shown very unusual features. During this epidemic, in the great majority of cases, actual contact with an actual case or with a child suffering from a definite, if mild, illness had been established. An attempt to restrict the disease to a limited area was therefore quite justified. If, however, sporadic cases occurred outside the defined area, the same measures of control that were at present being advocated might not be recommended.

DR. E. V. KEOGH said that at the Walter and Eliza Hall Institute he and his fellow workers had been successful in transmitting the virus in the present epidemic from cords of the first three fatal cases at the Children's Hospital to monkeys. Four monkeys had been used. They had obtained three typical positive reactions, and there had been only one failure to transmit. In the positive cases the monkeys had taken four, seven and eight days, respectively, to supply the evidence. It could be stated positively that this was a typical epidemic of poliomyelitis.

DR. JOHN DALE said that it was apparent that the greatest safety measure for children who were not affected was to keep them to themselves. They should be isolated and every precaution should be taken to limit exchange of sputum. The more the exchange of sputum could be limited, the less likely was it that their resistance would be overcome. This preventive measure was applicable not only to poliomyelitis, but also to all respiratory-borne disease. Dr. Dale was interested in the comments on the "red throat", and raised the question whether the virus affected the upper respiratory passages and not the olfactory epithelium only.

DR. JEAN MACNAMARA discussed the dramatic change in the epidemiology of this outbreak from that observed in epidemics in Victoria from 1925 to 1931. During that period the scattered incidence of cases had made it difficult to accept the doctrine of human carriage, for, in general, the cases had no more link between each other than had cases of broken bones. In this outbreak the connexion between patients had been obvious from the onset. The literature recording epidemiological studies showed a clear division into two groups: (i) as in the Swedish epidemics studied by Wickman, where human carriage was the obvious explanation; (ii) as in Kentucky recently where careful studies had led to the conclusion that an open mind must be maintained—that human carriage was difficult to accept as the explanation.

Dr. Macnamara suggested that the workers who had recorded these studies had been influenced by what they had seen and that both might be correct; that from time to time epidemics like this outbreak occurred, and at other times the type was like those that had occurred during the last twelve years. A hint as to the possible explanation might be available in veterinary medicine. Shope had shown that the virus of swine influenza acting alone caused a mild transient illness. Association of the virus with an organism was necessary to produce the severe, infectious disease "swine influenza". In Scotland, the Moredun workers had shown that the virus of louping ill caused the majority of sheep in a flock a mild transient general infection. The association of this virus with the virus of tick-borne fever seemed to increase its power to attack the nervous system. Louping ill had been a most

profitable disease to study for help in understanding poliomyelitis. Dr. Macnamara's first association with louping ill had been in laboratories where the disease was studied in laboratory animals—monkeys and mice. Because of its manifestations in these animals it was regarded as a disease of the nervous system caused by a neurotropic virus. In Scotland, however, where the disease was studied in its natural host, it was regarded and had been proved to be, in the majority of sheep, a general systemic disease, only going on to involvement of the nervous system in a small proportion of cases. It would be well to listen to the warning of the workers on louping ill, not to assume, because the virus followed certain paths in a laboratory animal, that the same thing happened in the natural host. These workers had also pointed out that if the virus of louping ill had been looked for in the blood of sheep at the stage in which the poliomyelitis virus had been looked for in the blood of children with pre-paralytic poliomyelitis, it would not have been found.

But it was there earlier, at the first rise of temperature. While the care with which the pathogenesis of monkey poliomyelitis had been studied merited respect, it might be irrelevant to human poliomyelitis. Dr. Macnamara could see no reason to change from the old conception of the human disease—a systemic disease in the majority affected, a proportion going on to involvement of the nervous system. She would not give up the conception that had satisfied Wickman, Caverley, Draper and scores of clinicians all over the world because of findings in an animal not naturally susceptible to the disease, which reacted to infection as the human did in the paralytic phase only. The monkey suffered from nothing to compare with the stage of general sickness. No one would describe the brief pre-paralytic illness of monkeys as a dengue state such as the Brisbane workers had noted in children. One had yet to read in reports of monkey infections descriptions of foul breath, coated tongue, injected throat, and general evidences of infection that were being sought in children at present. The present outbreak stressed the stage of general infection. As mentioned by Dr. Powell, fever had been high. Dr. Macnamara said that she would sound a note of warning in case the disease should revert to its old-fashioned type. She would hesitate to exclude poliomyelitis because high fever was absent. In the past, particularly in little children and in adults, the temperature had frequently been no higher than 37·2° C. (99° F.) throughout the illness. Alertness and desire to cooperate in the examination were symptoms in this outbreak that those present were familiar with; but the foul breath, coated tongue, flushed cheeks and injected throat were much more marked. In testing for the spine sign the most satisfactory method had been to ask the patient to lie on his side and curl around in order to kiss his knees. Dr. Macnamara recommended bribing the child with a penny. With high fever due to any cause it was unpleasant to sit up and bend the swimming head down to the knees, and discomfort might be confused with real pain. The side position saved the discomfort. In this outbreak, as in the past, Dr. Macnamara had found localized tremor to be the immediate herald of paralysis. She suggested that the head lag described by Dr. Powell and demonstrated in the film might be not a pre-paralytic sign, but evidence of paralysis of the flexor muscles of the neck, which had occurred frequently in this epidemic.

In a search for methods of control of this type of outbreak, Dr. Macnamara said that the most useful precedent was that which had occurred in Saugertes, in New York State, in 1916. It was found that isolation of paralysed patients had little effect. Every physician had guaranteed to isolate every patient with acute illness until diagnosis had been established. The epidemic had subsided soon after that plan had been put into operation; but the town had only 10,000 inhabitants.

Over thirty-nine winter outbreaks (mainly rural) of this disease had been recorded; the largest recorded had occurred in Vienna in 1908 with 145 cases. Dr. Macnamara suggested that it was not wise to remove tonsils during an epidemic like the present.

Dr. Macnamara did not agree with Dr. Powell that there was nothing the clinician could do to alter the course of the disease. As admitted by Andrews, even if serum was unable to save a cell that the virus had attacked, it might be possible to limit the number of cells attacked. In Victoria, in 1925, it had been said that it was impossible to diagnose poliomyelitis before paralysis had developed. Now early diagnosis was easy and common. It had been said, when an attempt was first made to obtain stocks of serum, that it would be impossible to obtain a sufficient supply; yet from 1926 to 1932 on no occasion had there been a shortage of fresh serum from proven paralytics for use in Victoria, though there had been four epidemics, and there had been enough for some to be sent to Tasmania and Brisbane. Dr. Macnamara admitted that it had taken energy and enthusiasm, but it had been done. Now they were told that because this disease might be different there was no point in trying to do anything. They were back to where they were, but had more opportunities of watching paralysis develop. Dr. Macnamara admitted that serum from patients in this outbreak would be more valuable than serum from patients affected in 1934; but why not obtain some fresh serum from old cases and use much larger doses than in the past? Dr. Macnamara said that she had given 150 cubic centimetres of serum to a child suffering from severe rapidly progressing infection at the stage of spine sign and tremor. It had not stopped the paralysis, which had killed the child; but the infection had been severe and the serum had been prepared in 1934 and refiltered. This was asking a good deal of stale material.

Dr. Burnet had referred to so-called controlled series of cases treated with and without serum. No controlled series had yet been carried out. Park naïvely admitted in his report that the two groups were not comparable, as a larger number of severe cases were included in the treated group, the mild cases being regarded as controls. One could not generalize about serum or about serum treatment in the absence of any unit or standard of potency. Results had varied with different batches with different dosages. As the Chicago workers had put it, the results had been directly proportional to the trouble taken. Until something better was offered, Dr. Macnamara considered it the plain duty of clinicians to use a method that had given good results in the past in affecting the general condition and in lessening the severity of paralysis.

A second method of influencing the disease had already been used this year by parents and medical practitioners in Melbourne. That was, protection from fatigue. The early cases in this outbreak had been very severe. In several the history had been obtained of exertion during the interval of apparent recovery. One child, aged six years, had gone six miles on her scooter forty hours before her death. It was possible that part of the lessened severity of the later cases might be attributed to the fact that at present any patient suffering from illness was put promptly to bed and kept there.

DR. CLIVE M. EADIE said that poliomyelitis was an infection of the posterior sinus area rather than the anterior. A condition such as coryza from the anterior sinuses should not be treated by sprays or douches, as the natural secretion had bactericidal powers. If any spread occurred, it was from the posterior nasal sinuses, and the object of treatment was to improve oxidation in that area by the provision of better ventilation and drainage; this could be done by inhalations alone; no sprays or douches should be used.

DR. STANLEY WILLIAMS, after congratulating Dr. Powell, said that he would like to make reference to one small point. He wished to emphasize the importance of the persistence rather than the appearance of neck stiffness and spine stiffness as a diagnostic feature in poliomyelitis. When one suspected *otitis media* or even pneumonia in the early stages of an acute febrile disturbance it was not unusual to find some neck or spine stiffness. If this persisted for as long as twenty-four hours it became of importance as a sign for distinguishing poliomyelitis from other conditions. It would then be justifiable to carry

out lumbar puncture. This procedure was not without danger to the patient when performed in the other conditions. Dr. Williams also expressed his satisfaction that it did not appear to be necessary to boil the milk as a preventive measure in poliomyelitis.

DR. F. L. TRINCA said that, in view of the apparent impotence of other remedies in the treatment of poliomyelitis, he made a plea for a remedy that did eradicate certain virus diseases. He referred to the preparation of sterile globulin known as "Edwenil". He recounted an experience with a lady who had been vaccinated and suffered great pain for four nights without being able to sleep. Within twenty-four hours of the injection of "Edwenil" the vesicles had become dry and she had been able to sleep comfortably. In speaking generally of the virus genus and of empirical remedies, analogies might not be applicable to poliomyelitis; but in their summary Topley and Wilson indicated that the mechanisms used against viruses and bacteria were practically identical. It had been proved experimentally that pseudo-globulin and euglobulin were effective in poliomyelitis and in vaccinia. Dr. Trinca stated that he would class "Edwenil" with insulin and "Salvarsan", and though it might not be curative always, in 90% of properly selected cases the result would be gratifying. In the 1935 epidemic of poliomyelitis in America Dr. Trinca stated that attenuated virus had been injected for protective purposes. Paralysis had occurred in the limb in which it had been injected. This fact supported the theory of axial spread.

PROFESSOR R. MARSHALL ALLAN, on behalf of the Branch, thanked the contributors to the discussion. He said that in his capacity as President he had received many inquiries from people about desirable measures to be adopted in the prevention of infection. He was glad to note that as a result of the present discussion he could tell inquirers that milk need not be boiled and that schools might not be closed. It could be taken as a sign of a new movement in health department circles that a special consultative committee had been formed to advise on poliomyelitis. This was an example of the way in which Dr. Featonby was going to get around him a group of expert advisers.

Dr. Powell, in reply to Dr. Burnet, said that it had been quite a noticeable feature how easily and clearly the children affected by poliomyelitis were able to breathe. If the volume of air entering by the nasal route had any relation to infection, it might be of interest to know that the children affected took in a greater volume by the nose than by the mouth. The people who were handling and treating the patients had, on Dr. Burnet's suggestion, been wearing masks made of eight thicknesses of gauze soaked in a solution consisting of equal parts of normal saline solution and glycerine, in the hope of minimizing the risk of infection. Dr. Powell said that he was glad to hear Dr. Macnamara say that cases of less severity could be expected in the future course of the prevailing epidemic. He agreed with this view and thought it probable that this phase had already commenced, because he certainly thought that the cases he had seen that week had been harder to diagnose. He had not overlooked the fact that he was seeing them earlier in the illness; but it was true that he had not seen one that was easy to diagnose that week. The presence of neck stiffness in the absence of head retraction recalled a case in which these features had been so prominent that tetanus had to be excluded from the differential diagnosis. Another point he had noticed was that the amount of back rigidity was out of proportion to the small amount of head retraction. In meningitis with an equivalent amount of back rigidity he would have expected the patient to have been opisthotonic. Another point in the differential diagnosis from early meningitis was that patients with poliomyelitis were astonishingly lucid and altogether free from the tendency to the mental impairment found in the early stage of meningitis. Referring to Dr. Macnamara's remarks on the use of serum, Dr. Powell mentioned that in December, 1936, he had written to Dr. C. F. McKhann, of Boston, asking him for the present opinion in Boston concerning the value of serum. He felt it would be sufficient to quote one sentence from the reply: "We still use convalescent serum in pre-paralytic poliomyelitis, but we have no faith in it." This striking statement was from an experienced man who possessed a sound clinical judgement. Dr. Powell said that he intended to test the efficacy of serum from abortive cases in the present epidemic. Experimental work had been done showing that serum from abortive cases was 150 times and serum from non-paralytic cases 75 times as potent as serum from paralytic cases. He was perfectly prepared to be disappointed, and was particularly anxious that no newspaper publicity should be given to this aspect of the treatment. Dr. Powell considered that the suggestion of Dr. Williams about keeping the patients under observation for twenty-four hours before reaching a decision was open to criticism. Nevertheless there was no need to be panicky, and in certain cases it might take forty-eight hours' observation in the home before the diagnosis could be made. In such cases it was of the utmost importance that the suspect should be isolated from the other people in the house. At the moment they were powerless to stop infection promptly, as the damage was already done. Dr. Powell stated that he had not had any experience of "Edwenil" in the treatment of poliomyelitis and he was perfectly prepared to see a trial of it. One could not argue against success; but Dr. Powell wondered whether in five or ten years' time Dr. Trinca would be prepared to say the same things about it, or whether "Edwenil" would be just another preparation that would not stand the test of time.

A MEETING of the Section of Medicine of the New South Wales Branch of the British Medical Association was held at the Robert H. Todd Assembly Hall, 135, Macquarie Street, Sydney, on April 22, 1937, DR. H. J. RITCHIE, President of the Section, in the chair.

The Functional Pathology of Anæmia.

PROFESSOR C. G. LAMBIE read a paper entitled "The Functional Pathology of Anæmia". This paper was a *résumé* of the articles by Professor Lambie that have appeared in the issues of August 21, 28 and September 4, and of the paper in this issue at page 423.

DR. C. G. MCDONALD said that he had unfortunately arrived late; but he doubted, if he had arrived early, whether he would have been able to understand portions of Professor Lambie's lecture any better. He admitted that he had been unable to follow some of Professor Lambie's diagrams and complicated formulæ. He wondered if all others present could grasp with facility an argument on biochemical lines; he confessed that he was unable to do so himself. Listening to such an argument produced in him a pronounced sense of inferiority. This sense of inferiority had been aggravated greatly when Professor Lambie had shown dissociation curves "expressed logarithmically". He had felt relief when the lecturer had turned from the maze of biochemical details to the discussion of the clinical features of anæmia. He agreed that Professor Lambie had explained these very fully. He was relieved to find that the ferment calcium anhydrase came to the rescue of the patient at a critical stage. While all might not subscribe to Professor Lambie's explanations of all the symptoms, they would agree that no more complete description could be given. Dr. McDonald was glad that the lecturer had emphasized the pulmonary origin of dyspnœa in congestive cardiac failure and had contrasted it with the cause of anæmia. Dr. McDonald remembered that a colleague some years previously had asked him what was the cause of dyspnœa in congestive cardiac failure, and he had replied that it was oxygen lack; but his colleague had declared that pulmonary congestion was the cause. At the time Dr. McDonald had been doubtful of his friend's seriousness. But it was becoming more and more obvious that the cause of cardiac dyspnœa was essentially vagal stimulation caused in some way by the pulmonary congestion, probably by means of oxygen lack in the pulmonary circulation. The question had been put to him many years ago: Why, if stimulation of the

respiratory centre by oxygen lack in the general circulation was the cause of dyspnœa, was there not much more severe dyspnœa in anæmia? Why did a patient suffering from pernicious anæmia and having but one million red cells to each cubic millimetre of blood with the gross reduction in oxygen-carrying hæmoglobin which such anæmia entailed, lie in bed without gross dyspnœa if oxygen lack was the main cause of breathlessness?

Dr. McDonald concluded by expressing the hope that Professor Lambie's paper would appear soon in print. He looked forward with pleasure to a careful reading of its contents.

DR. G. C. WILLCOCKS said that he was in much the same position as Dr. McDonald in trying to discuss Professor Lambie's paper; but perhaps he had understood a little more of it than Dr. McDonald. He had been especially interested in Professor Lambie's explanation of the symptoms and he had been glad to find there were still some that could not be explained. Professor Lambie had stated that the pulmonary condition was the cause of the dyspnœa in cardiac failure. If this was so, was oxygen administration of any value? The teaching had been that it was; but Dr. Willcocks had never seen it do much good. Professor Lambie had mentioned that weakness was the earliest symptom of anæmia. But Dr. Willcocks had observed many people who were able to get about for a long time despite very severe anæmia. Before the introduction of blood transfusion and the intramuscular injection of liver it had been a common experience to get patients with pernicious anæmia too late to save them. This was an example of the body's powers of compensation; but it was difficult to understand how a patient could get about with no more than one million red cells per cubic millimetre of blood. In conclusion, Dr. Willcocks thanked Professor Lambie for his learned paper and promised that, like Dr. McDonald, he would study it carefully when it was published.

DR. H. J. RITCHIE said that he was sure all those present had enjoyed Professor Lambie's paper even if they had not been able to absorb it all. He should like to say that they owed Professor Lambie a very deep debt of gratitude for his able and scholarly address. Professor Lambie had mentioned the absence of cardiac pain in anæmia. Dr. Ritchie was inclined to think, from his own experience, that various forms of cardiac pain were not uncommon in anæmia, even when the factor of sclerosis was excluded. As a general proposition, it was of interest that Professor Lambie had demonstrated biochemically what all found out clinically, namely, that anæmia might become very severe before pronounced symptoms of distress appeared, but that once such symptoms appeared the patient's downhill progress became very rapid. Once the hæmoglobin value fell below 50% the patient's condition was apt to become rapidly worse, and once it fell below 30% the patient was in danger of dissolution. Dr. Ritchie concluded by expressing the wish that Professor Lambie would elaborate his remarks on cardiac pain.

Professor Lambie, in reply, said that he owed the meeting an apology for exceeding the time allotted to him for the reading of his paper; but he did not intend to apologize for an address that some of those present were incapable of understanding. To present to people something they could not understand was one way of stimulating them to learn. In the teaching of students it was a good thing to lift them off their feet sometimes; then when they came down they would stand more firmly. In order to understand the common phenomena of disease it was necessary to study physiological and biological phenomena. Any knowledge that did not probe to those depths was superficial.

In reply to Dr. Willcocks, Professor Lambie said that oxygen administration was of little value in congestive cardiac failure unless there were pulmonary complications. When, for any reason, the diffusion of oxygen was impaired, oxygen therapy could do good. If the saturation of oxygen in the blood was normal, the addition of oxygen was of little use; but in pulmonary disease, when the blood was poisoned by an insufficiency of oxygen, it could be administered with advantage. Dr. Willcocks had mentioned the ability of patients to get about despite severe anæmia. Professor Lambie said that this was a point he had stressed in his paper. The patient could do this because of the phenomenon of adaptation and because of the slowness of the production of the anæmia. The same thing occurred in ascent to high altitudes. But the patient with anæmia was in a better position than the person going to a high altitude, because, although a hæmoglobin value of 50% was equivalent to the effects of a height of 20,000 feet, a person going to a high altitude did not increase his circulation rate so quickly, owing probably to the effects of acapnia—washing out of carbon dioxide owing to sensitization of the respiratory mechanism by oxygen lack—upon the capillaries, and adaptation in this condition was difficult. But in anæmia acapnia did not occur. In anæmia also the viscosity of the blood was diminished, whereas in mountain sickness the red blood cells were increased in number and the viscosity was increased. In anæmia of insidious onset there was also plenty of time for adaptation to low oxygen tissue tension. The anæmic patient was therefore better able to adapt himself to the altered conditions.

In reply to Dr. Ritchie, Professor Lambie said that in the reports on cardiac pain in anæmia most observers stated that they were dealing with patients suffering from arterial disease. As Professor Lambie had pointed out in his paper, anæmic patients with arterial disease would be more liable to cardiac pain than persons with normal blood. There might be a certain degree of sclerosis of the coronary vessels without any observable sclerosis of the radial arteries. He had been glad to hear Dr. Ritchie summarize the important points of his paper. The reason why dissolution was imminent when the hæmoglobin value fell to 20% was that compensation was then impossible. The flow of blood could not be increased to such an extent as to make up for the loss of hæmoglobin. The coronary blood supply had to be enormously increased in anæmia. There would be a limit to this supply, and when this limit was reached the heart was bound to give out.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal Alexandra Hospital for Children, Sydney, on April 22, 1937. The meeting took the form of a series of demonstrations by members of the honorary staff. Part of this report appeared in the issue of September 4, 1937.

Cerebellar Tumour.

DR. T. Y. NELSON showed a girl, aged twelve years, who had been admitted to hospital on October 12, 1936, with a history of vomiting of more than four months' duration; the symptoms would disappear for a few days, after which vomiting would occur again. Occipital headache and blurring of vision had been present for about three weeks and vomiting had been constant for one week. Examination of the cranial nerves revealed no abnormality except for slight blurring of the optic disks; but the presence of slight ataxia nystagmus and adiadochokinesia, especially on the right side, indicated a cerebellar disturbance, although there was very little alteration of gait. Examination of the skull by means of X rays revealed no abnormality.

On November 17, 1936, ventricular estimation was carried out, and forty cubic centimetres of cerebro-spinal fluid were removed from the right ventricle and twenty cubic centimetres from the left. No radiographs were made, as it seemed evident that a symmetrical internal hydrocephalus was present. At operation, performed on November 24, a large, solid tumour was disclosed, which separated the two cerebellar hemispheres; it had a cystic prolongation extending down the spinal canal, past the level of the atlas. The arch of the atlas was removed and the cystic portion of the tumour excised; no attempt was made to remove the main part of the tumour. Dr. Nelson said that since the operation the child had had two full

courses of deep X ray therapy. She had remained well, and, judging by her excellent response to the treatment, it seemed likely that the tumour was a medulloblastoma.

Dr. Nelson's next patient was a girl, aged ten years, who appeared to be suffering from cerebellar tumour, and who had been admitted to hospital on May 15, 1936, with a history of vomiting of twelve months' duration. Soon after the commencement of the illness an internal strabismus of the child's left eye was noticed. Frequent headaches, mainly frontal, had been a feature of the illness throughout, and diplopia had been observed during the two weeks prior to the patient's admission to hospital. Examination of the cranial nerves revealed no abnormality, and in spite of the diplopia no paralysis of the ocular muscles was present; papilœdema was present in each eye to the extent of four diopters. The fields of vision and the labyrinthine function were normal. The only cerebellar symptom was a tendency to fall to the left when the child stood in the tandem position; the gait was normal.

X ray examination of the skull revealed widening of the sutures due apparently to a chronic rise of the intracranial tension, and some minute calcified nodules on the left side, probably close to the cortex. Ventricular estimation made on June 16, 1936, revealed symmetrical dilatation of both ventricles; the cerebellum was at once explored. No tumour was found; but the arachnoid appeared to be denser than normal and some thickened areas were broken down. Later on, in view of the lack of positive findings, a ventriculogram was made, and the presence of symmetrical hydrocephalus was confirmed. At the time of the meeting the child was well and the papilœdema had subsided.

Cerebral Abscess.

Dr. Nelson's next patient was a child, aged three years, who was suffering from a metastatic cerebral abscess. The patient had been admitted to hospital on December 18, 1936, with pneumonia. An empyema formed and was drained on January 16, 1937, by means of a tube inserted through the eighth right intercostal space; streptococci and pneumococci were grown in culture from the pus. On January 27 a rib section was performed and the cavity was drained.

Progress was uneventful until February 15, when the sinus had practically closed; the following day flaccid paralysis was observed in the child's left arm. Two days later examination revealed elevation of the temperature, flaccid paralysis of the left arm and exaggeration of the deep reflexes of both legs, with clonus of the ankle on both sides, especially the left. No disturbance of sensation was observed, and the abdominal reflexes were present and the plantar response was flexor. The child was kept under observation for some days, during which time the signs remained the same; no papilœdema appeared and the number of cells in the cerebro-spinal fluid did not increase. Facial weakness was then observed on the right side. Dr. A. W. Campbell saw the child and made a diagnosis of cerebral abscess below the Rolandic area on the right side.

The motor area was explored by operation. On March 9, 1937, pus was encountered at a depth of five centimetres from the cortex, and about four cubic centimetres were aspirated. The abscess was drained by means of two rubber tubes, inserted by being first fitted over a brain needle, which was then introduced into the cavity. Drainage was very slow, and by March 21 pronounced *hernia cerebri* was present: this was controlled by means of daily lumbar puncture. On March 28 the abscess was again aspirated and drained; thirty cubic centimetres of foul-smelling pus were removed and a rubber drain was inserted in the cavity. Dr. Nelson said that the child's progress had been satisfactory. At the time of the meeting a small sinus remained and the left arm was slightly weak.

DR. NORMAN MEACLE showed a girl, aged eight years, who had been admitted to hospital on August 5, 1936, with a history of having had chicken-pox three weeks previously, and of having had a discharging ear one year previously. The tonsils and adenoids had been removed. On admission to hospital the child complained of earache in the right ear, and a swelling was visible behind the ear. Fever was present, but no vomiting. Schwartze's operation was performed the same day. Pus was found in the antrum, and a small perisinuous abscess was seen when the lateral sinus was exposed. Granulations were found on the *dura mater* when the middle fossa was opened.

On August 17 the child was very drowsy and difficult to rouse; the pulse rate was between 60 and 70 per minute; the temperature was normal. Lumbar puncture was performed and clear fluid was obtained under pressure. During the afternoon of the same day the child's drowsiness increased; the limbs were flaccid; the reflexes were active on the right side and absent on the left side. Facial paresis and ptosis of the eyelid were observed on the left side. The plantar reflex was extensor on the left side and flexor on the right side. Operation was performed on the same evening, and a brain needle was inserted through the *dura mater* of the middle fossa. Pus was encountered at a depth of 1·25 centimetres (half an inch); a considerable amount was evacuated and a rubber drainage tube was inserted in the wound and left in. During the operation the pulse rate rose to 120 per minute. On August 18 the child was completely conscious and her condition was good; there was no paresis. The wound was dressed every day and the tube was shortened every second day; the amount of discharge was decreasing. On September 23 the child was discharged from hospital quite well; the wound had healed.

Dyslalia or Immature Speech.

DR. D. W. H. ARNOTT said that dyslalia was a condition in which the normal "baby speech" was abnormally prolonged into childhood. It consisted of an inability to pronounce properly certain consonants, especially "g", "l" and "r". It was often associated with, but did not by itself indicate, mental deficiency. Unless it was associated with a fairly severe grade of mental deficiency, as a general rule it responded rapidly to speech training.

Dr. Arnott showed several patients to illustrate the condition, the first being a girl, aged eight years, whose birth and childhood development had been normal. She had been born with one tooth, which fell out when she was six weeks of age, and her palate had a very high vault. Her intelligence quotient was 88%. Dr. Arnott thought that the three last factors indicated a defect due to faulty development. The child's speech was very babyish, and the fact that it had remained so up to her eighth year suggested a serious disturbance of the speech centre. Dr. Arnott thought that, although the speech would improve with training, some speech defect would remain.

Dr. Arnott's three next patients were children from the same family, a boy, aged ten years, and two girls, aged respectively seven years and nine months and five years and six months. Both parents were epileptic and alcoholic, and the children were all State wards; one sister was an epileptic and in the Newcastle Mental Hospital, and one brother had died of "water on the brain". Of the three children shown, the boy had an intelligence quotient of 75%; his speech was very babyish, and in addition many of the words were cut short, while in others the final syllable was accentuated. Although his prepared speech work had improved, his spontaneous speech had not, and Dr. Arnott thought that, on account of the mental deficiency, the prognosis was not very good.

The speech of the elder of the two girls was practically the same as the boy's, but not quite so bad. The child's intelligence quotient was 87%, and her dulness had been accentuated by two epileptic fits which she had had. Dr. Arnott considered that before the fits her intelligence was probably very close to normal, and that her speech defect had been perpetuated by the child's imitating her brother. It was not likely that she would be completely cured.

The younger girl had had convulsions as a baby, but in spite of this was the brightest of the three children, having an intelligence quotient of 103%. The fact that her speech was the same as that of the other children was probably due to imitation. On account of the child's normal mind the prognosis was better, and this was borne out by

the fact that she was the only one of the three who had made progress in spontaneous speech as a result of speech training.

Tuberculous Disease of the Knees.

DR. SHEDDEN DAVIS showed a boy who had been admitted to hospital at the age of four years suffering from a tuberculous lesion of the left knee. This diagnosis had been finally accepted despite the absence of any reaction to von Pirquet's test or to the hypodermic injection of old tuberculin. Treatment consisted of rest in a Hamilton splint, with extension of the limb, at first, on account of the night starts. Graduated doses of bacillary emulsion tuberculin were given by injection. The result might be classed as a complete cure.

Dr. Davis also showed X ray photographs of another smaller boy, who could not be shown at the meeting. This child was suffering from tuberculosis of both knees, which had been progressive for many months despite rest in a Hamilton splint and tuberculin injections. In the right knee the disease was confined to the soft tissues and synovial membrane, with consequent slight dislocation of the tibia backwards. The skiagrams of the left knee showed a very progressive lesion of the lower end of the femur, which, however, had left the knee freely mobile, with very little tenderness or swelling, although the leg was extremely wasted. A short time only before the meeting had there been any clinical evidence of arrest of the tuberculous process.

Lymphadenoma.

Dr. Shedden Davis also showed a patient suffering from lymphadenoma. This girl, aged eleven years, had noticed a small swelling of the glands of the right side of the neck in December, 1936. She had been admitted to hospital in February, 1937, with two or three large and hard, yet discrete, glands in the right posterior triangle of the neck. Glands were palpable in both axillæ, and there were some crepitations at the base of the right lung. Examination of the blood revealed only a diminution of the hæmoglobin value. X ray examination revealed a large right-sided intrathoracic mass. There was no reaction to the Wassermann test. Biopsy of the glands in the neck confirmed the diagnosis of lymphadenoma. In the middle of March a course of nine exposures to deep X rays was completed, and X ray examination then revealed that the mass had shrunk by about 80%. The child was readmitted to hospital on April 19, but no advance in the condition was observed clinically. X ray examination perhaps revealed some increase in the thoracic tumour. Dr. Davis said that it was proposed to give the child a further course of deep X ray therapy. The interesting feature of this case was the absence of symptoms from the beginning, despite the interference with the thoracic contents by a large mass.

Osteochondritis Deformans.

Dr. Shedden Davis also showed a child, aged five years, who had been admitted to hospital suffering from a mild arthritis of the left ankle, which had subsided after treatment by salicylates. Routine examination by X rays disclosed a symptomless *osteochondritis deformans* of the first cuneiform bone on each side. Dr. Davis proposed to treat this with strapping, to support the arch, relieving if possible some of the pressure on the cuneiform bone; plaster of Paris could be used later if the bones became deformed.

Osteogenic Sarcoma of the Right Femur.

Dr. Shedden Davis's next patient was a girl, aged ten years, suffering from osteogenic sarcoma of the right femur. In December, 1936, a lump had been noticed at the inner side of the lower end of the right femur. X ray examination made early in January, 1937, revealed no abnormality. At operation, performed on February 3, bare bone was seen, but no pus. X ray examination on February 16 revealed an erosion of the bone; whether this was osteomyelitis or sarcoma was not decided. A further incision was made on March 3, and a friable vascular tumour, with some bone destruction, was seen; a specimen was sent for examination to the Board of Health, the report being "osteogenic sarcoma". The child was then sent into the Royal Alexandra Hospital for Children, and a course of deep X ray therapy was commenced at once. When this was complete, on April 9, disarticulation of the limb at the hip joint was undertaken, the child being given paraldehyde *per rectum* and an intraspinal anæsthetic, after which very light ether anæsthesia was induced. Despite these precautions, the shock was very severe, although the child quickly picked up afterwards.

Dr. Tidswell's notes as to the tumour itself were as follows:

> There was a large tumour superficial to the femur, apparently springing from the periosteum. It was fifteen centimetres in length, thirteen centimetres wide at the centre, and six centimetres deep from the skin surface to the femur. The stretched area of the biopsy scar was visible on one side, beneath which the tumour felt much harder than in other areas.
>
> The cut surface showed a lobulated appearance, with irregular areas of hæmorrhage and degeneration. The muscle tissue above the well-defined upper border of the growth was fibrosed and yellow and tough to cut. The marrow cavity of the femur deep to the tumour and in cross section above it appeared to be normal.

Dr. Davis said, with regard to the prognosis, that Binnie held that "the results of amputation are vile". Another writer had reported on twenty-three patients suffering from osteogenic sarcoma and from sarcoma of the long bones, of whom eleven were dead and only six alive and well, a percentage of 26. The period of time covered by the report was not stated.

Umbilical Fistula.

Dr. Davis also showed a boy, aged six months, who had suffered from a discharging umbilicus after separation of the cord. The discharge was thick and greenish brown in colour, and intermittent hæmorrhage occurred. The tissues surrounding the umbilicus were indurated and excoriated. At operation, performed on April 14, 1937, a persistent Meckel's diverticulum opening into the umbilicus was found. This was tied and removed, and the urachus was also ligated.

Pathological Exhibits.

DR. F. TIDSWELL showed a number of specimens illustrating various pathological conditions in infants. Speaking of umbilical sepsis, he said that infection implanted on the raw umbilicus might reveal itself by superficial and visible reaction, or might track deeply and invisibly along channels leading from the umbilicus. The umbilicus could be described as a roughly circular, decorticated area, within which were the openings of two ducts, the vitelline duct (intestinal umbilicus) and the allantoic duct (urinary umbilicus), and of several blood vessels (the hypogastric arteries and vitelline veins). In normal circumstances, these structures were obliterated and rendered functionless at birth, and they atrophied from disuse very soon afterwards. At times their existence was prolonged, and they became pathways along which infection spread inwards. Most commonly the course was upwards along the vitelline veins, with resulting abscesses of the liver; more rarely a patent urachus allowed downward progress, the result being pelvic inflammation. Purely local infection resulted in umbilical abscess or fistula, but peritonitis and general sepsis might follow. Dr. Tidswell said that it was unfortunate that the deeper lesions could occur in the absence of external signs; the umbilicus might be quite healthy in appearance. Moreover, the infection might be latent or slow and might not reveal itself until after the usual period of observation. It could be assumed that umbilical sepsis was a corollary of maternal sepsis, and would probably be minimized by the institution of precautionary measures against that condition.

Dr. Tidswell then referred to the condition of persistent vitelline duct. He said that the embryonic duct might

persist as a sac attached to the umbilicus (omphalic fistula), or a communication between the umbilicus and the bowel (omphaloenteric fistula), or a sac attached to the intestine (enterocyst or Meckel's diverticulum).

Speaking of congenital heart disease, Dr. Tidswell said that it was dependent on anatomical defects; those defects which were incompatible with individual existence occurred in stillborn children and were not represented in the series of specimens he had prepared. Dr. Tidswell then showed some exhibits illustrating conditions which admitted of some period of post-natal survival in spite of the difficulties caused by interference with the normal functions of the heart. Most of the specimens illustrated some imperfection of one or other of the septa which normally separated the chambers of the heart or of the valves between them; the result of the imperfection would be a shunt or misdirection of the blood within the heart. Dr. Tidswell pointed out that many of the exhibits revealed the presence of more than one defect, and it could be said that this was the rule.

Correspondence.

DENTISTRY IN THE COUNTRY.

SIR: Your editorial article on "Dentistry in the Country", in your issue of August 21, 1937, prefers two charges against the dental profession.

(1) "We have yet to learn that the dental profession has formulated a scale of fees that may reasonably be called 'intermediate'."

(2) "Medical treatment is available for the poor and needy, surely some attempt can be made to provide dental treatment, at least for those who urgently require attention."

In the absence of an accurate definition of the word "intermediate", we assume you mean a reduction of ordinary fees charged.

For the dental profession to provide an intermediate scale of fees presumes that the individual practitioner can carry the burden of a lower fee basis, or alternately, provide a lower standard of service, in conformity with the reduced scale of fees. In the interests of public health, neither possibility is desirable.

The principal difficulties confronting the dental profession in a consideration of "intermediate" fees is the time factor and the high tariff-laden cost of materials and equipment. The time required for any operative service limits the number of patients that a dentist can attend daily.

The problem of providing efficient dentures for the basic wage earner and his family is definitely dependent on the time factor and costs, and cannot be complacently solved with editorial simplicity.

Although no easy economic solution of this complex difficulty has been attained, it is incorrect to assume that the responsible bodies are not seriously exercised on this important health issue.

Your second statement infers that no satisfactory or serious attempt has been made to solve the problem of treatment for the needy members of the community, particularly in the country districts.

It may be emphatically stated that the Hospitals Commission has energetically attacked the problem in so far as the funds available have permitted.

The Australian Dental Association has cooperated completely with the Commission, and members of the Association in country districts are voluntarily manning clinics in hospitals. A very carefully organized system is in operation, which is only limited by the Government subsidy obtainable.

Already the dental train clinic for the treatment of adults and children in far-lying districts has proved a successful experiment, and another will shortly come into operation.

The Australian Dental Association is fully aware and appreciative of the fact that many members of the profession practising in country towns where no clinics exist, provide a great deal of gratuitous service to necessitous cases for which they neither seek nor require recognition. This treatment entails a direct expense to the dentist.

Your editorial certainly implies that the dental profession is not aware of the difficulties of combating dental disease, or making any serious constructive contribution towards this important aspect of public health and national efficiency.

On behalf of the Australian Dental Association, I can assure you that our policy has been energetically pursued, but as in the progress of medicine spectacular results are not achieved by public criticism of an ethical professional body.

The fact that your editorial produced headlines in the daily Press, such as "The Doctor Hits at Dental Fees", is most unfortunate and not conducive to that happy feeling of cooperation that exists between the two bodies in their endeavour to promote the interests of public health.

The Australian Dental Association, in its warm respect for the British Medical Association, would hesitate to publish any article in its journal suggestively critical of medical services to the community, and I am convinced this policy is above reproach.

Yours, etc.,
J. V. HALL BEST,
President.

Australian Dental Association,
New South Wales Branch,
137, Macquarie Street,
Sydney,
August 30, 1937.

Obituary.

EDWARD FAIR PERRY.

WE regret to announce the death of Dr. Edward Fair Perry, which occurred on August 10, 1937, at Goondiwindi, Queensland.

JOSEPH KEITH PARK.

WE regret to announce the death of Dr. Joseph Keith Park, which occurred on August 23, 1937, at Goodna, Queensland.

FELIX MEYER.

WE regret to announce the death of Dr. Felix Meyer, which occurred on August 31, 1937, at Armadale, Victoria.

ALEXANDER HAMILTON RUTHERFORD.

WE regret to announce the death of Dr. Alexander Hamilton Rutherford, which occurred on September 2, 1937, at Sydney, New South Wales.

Corrigendum.

OUR attention has been drawn to an error that occurred in the journal of August 28, 1937, at page 364. Dr. A. P. Derham is reported to have stated that "Luminal" came into general use in Melbourne in 1932. This should read "1922".

Books Received.

PSYCHOANALYSIS EXPLAINED, by D. R. Blitzsten, with an introduction by A. A. Brill, M.D.; 1937. London: George Allen and Unwin. Crown 8vo, pp. 76. Price: 3s. 6d. net.

MODERN PSYCHOLOGY IN PRACTICE, by W. L. Neustatter, B.Sc., M.B., B.S., M.R.C.P., with a foreword by R. D. Gillespie, M.D., F.R.C.P., D.P.M.; 1937. London: J. and A. Churchill Limited. Large crown 8vo, pp. 314. Price: 10s. 6d. net.

THE ELEMENTS OF MEDICAL TREATMENT, by R. Hutchison, M.D., LL.D., F.R.C.P.; Third Edition; 1937. Bristol: John Wright and Sons Limited. Crown 8vo, pp. 198. Price: 5s. net.

Diary for the Month.

SEPT. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 15.—Western Australian Branch, B.M.A.: Branch.
SEPT. 21.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 22.—Victorian Branch, B.M.A.: Council.
SEPT. 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 24.—Queensland Branch, B.M.A.: Council.
SEPT. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 30.—South Australian Branch, B.M.A.: Branch.
SEPT. 30.—New South Wales Branch, B.M.A.: Branch.
OCT. 1.—Queensland Branch, B.M.A.: Branch.
OCT. 5.—New South Wales Branch, B.M.A.: Council (Quarterly).
OCT. 6.—Victorian Branch, B.M.A.: Branch.
OCT. 6.—Western Australian Branch, B.M.A.: Council.
OCT. 7.—South Australian Branch, B.M.A.: Council.
OCT. 7.—Tasmanian Branch, B.M.A.: Council.
OCT. 8.—Queensland Branch, B.M.A.: Council.
OCT. 12.—New South Wales Branch, B.M.A.: Organization and Science Committee.
OCT. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments.

Dr. J. B. Lewis has been appointed Honorary Consulting Ophthalmologist at the Adelaide Hospital, Adelaide, South Australia.

* * *

Dr. R. I. Campbell has been appointed Government Medical Officer at Lithgow, New South Wales.

* * *

Dr. G. V. Hickey has been appointed an Official Visitor to the Willowburn Mental Hospital, pursuant to the provisions of *The Insanity Acts*, 1884 *to* 1935, of Queensland.

* * *

Dr. A. Gild has been appointed Surgical Registrar at the Adelaide Hospital, Adelaide, South Australia.

* * *

Dr. L. A. Langley has been appointed a Medical Officer in the Department of Mental Hygiene of Victoria, under the provisions of the *Public Service Act*, 1928, of Victoria.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xvi to xviii.

AYR HOSPITALS BOARD, AYR, QUEENSLAND: Junior Medical Officer.
DIRECTOR-GENERAL OF PUBLIC HEALTH, SYDNEY, NEW SOUTH WALES: Honorary Radiologist.
THE KARAMEA MEDICAL ASSOCIATION OF NEW ZEALAND: Medical Officer.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Honorary Ear, Nose and Throat Surgeon, Resident Medical Officer.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Surgeons.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135 Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178 North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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